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Spinal epidural abscess with gadolinium-enhanced MRI: serial follow-up studies and clinical correlations

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Abstract. We reviewed serial MRI with and without gadolinium-DTPA in eight patients with spinal epidural abscess and correlated the findings and the clinical manifestations. In four patients, diffuse abscesses spanned four vertebral bodies or more; the others had focal abscesses associated with osteomyelitis and/or diskitis. In three of the four patients with diffuse abscesses, MRI (NCMRI) showed diffuse encasement of the subarachnoid space. Contrast-enhanced MRI (CEMRI) demonstrated linear enhancement surrounding unenhanced pus. In the four patients with focal abscesses, CEMR delineated the inflammatory process more clearly than NCMR. On follow-up studies, decrease in abscess size and better visualization of the subarachnoid space correlated with clinical improvement in both diffuse and focal abscesses. Despite clinical improvement, contrast enhancement persisted in the disk or epidural space of three patients, and was thought to represent chronic granulomatous change or postsurgical scar. CEMR is very valvable for the initial diagnosis of an epidural abscess, particularly if it involves lengthy segments. During follow-up, CEMR can document responses to therapy, and provide information for determining appropriate treatment.

Key words: Spinal infection – Abscess – Magnetic resonance imaging

Spinal epidural abscess (EA) is uncommon, but its incidence seems to be increasing. Untreated, this disease leads to paraplegia, quadriplegia, or even death. Survival and prevention of serious neurologic deficits relate to the rapidity with which appropriate decompression and antibiotic therapy are instituted; prompt diagnosis is therefore critical [1–5]. While myelography with CT has been the study of choice in patients suspected of having an EA, several reports recently addressed the usefulness of MRI [6, 7]. However, the diagnosis is not always possible on unenhanced images (NCMRI) [6]. Gadolinium-diethylenetriamine penta-acetic acid (Gd-DTPA) can be a value adjunct to NCMRI by enhancing actively inflamed tissue [8]. Post et al. [9] described the significant advantage of gadolinium enhanced MRI (CEMRI) in the diagnosis of spinal epidural abscess [9].

We describe features of CEMRI in 8 patients with EA, which have not been fully described, and evaluate correlations between follow-up MRI and the clinical manifestations.

Materials and methods

Sequential MRI of the spines of 8 patients with spinal EA admitted from April 1989 to February 1991 were studied retrospectively. The findings were compared with the clinical manifestations and pathologic data. The diagnoses was established in six patients at decompressive surgery, at open biopsy in another, and by aspiration of pus in the last. There were 3 males and 5 females, whose ages ranged from 9–70 years, average 45 years. They included three intravenous drug users; four had diabetes mellitus, three a history of back trauma, one had undergone hemodialysis, and one a recent dental extraction. Three patients presented primarily with back or neck pain without neurological deficits, whereas five patients sought medical attention because of symptoms of cord compression. The bacterial pathogens were identified in all patients: *Staphylococcus aureus* in five, *Streptococcus viridans,* group A streptococcus and pseudomonas in one each. Following Post et al. [6], we classified infections spanning four vertebral bodies or more as diffuse epidural abscesses. These were seen in four patients, two of whom had panspinal abscesses, one a thoracic and another a lumbar abscess. In the other four patients the infection, localized to four or less vertebral body segments, was classified as a focal epidural abscess; two were in the cervical region, one was thoracic and the other lumbar.

Initial MR images were obtained prior to treatment in all patients; the interval from the onset of symptoms ranged from two days to six months. Serial MRI was obtained 1–6 times over periods of 7 days to 6 months. MRI was performed using 1.5T units equipped with standard surface coils. For NCMRI, T1-, proton density- and T2-weighted sagittal images were obtained, with a matrix of 192 × 256 or 256 × 256,

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and with 4–5 mm thick sections supplemented by 5 mm thick axial sections. T1-weighted spin echo (SE) sequences were performed with 450–650/17–20/2 [TR(ms)/TE(ms)/number of excitations]. Proton density- and T2 weighted sequences were obtained with SE 2000–3500/25–45, 90/1. Cardiac-gated images were obtained in the thorax. Gd-DTPA, 0.1 mmol/kg, was injected intravenously. T1-weighted images were obtained in the sagittal and axial planes immediately after injection. In the diffuse abscesses, the levels clinically suspected of harbouring the primary infection were examined using NC- and CEMR, followed by CEMR covering the remaining levels. In selected cases, additional axial gradient echo images (TR/TE/flip angle = 300/19/30°) were obtained.

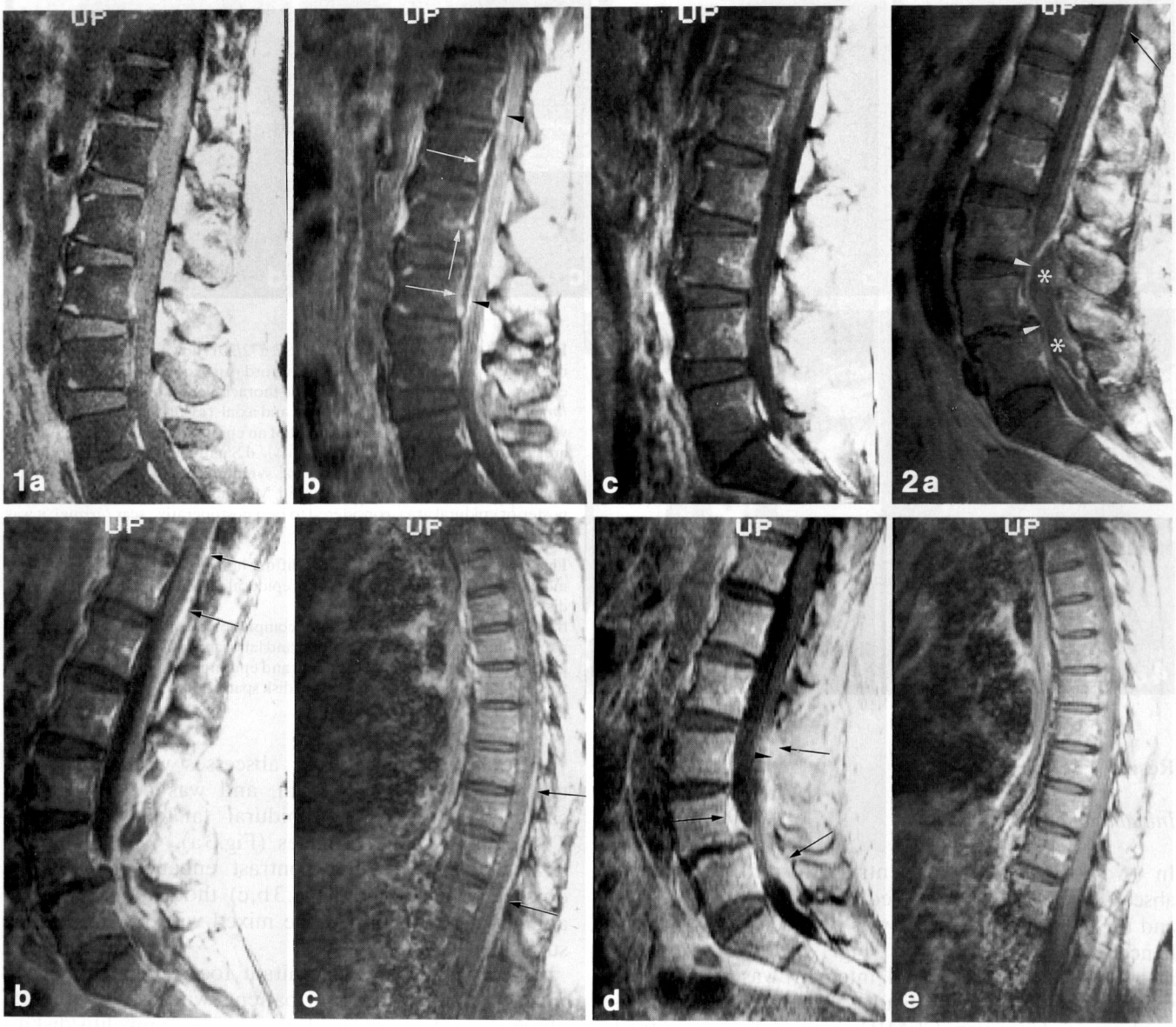
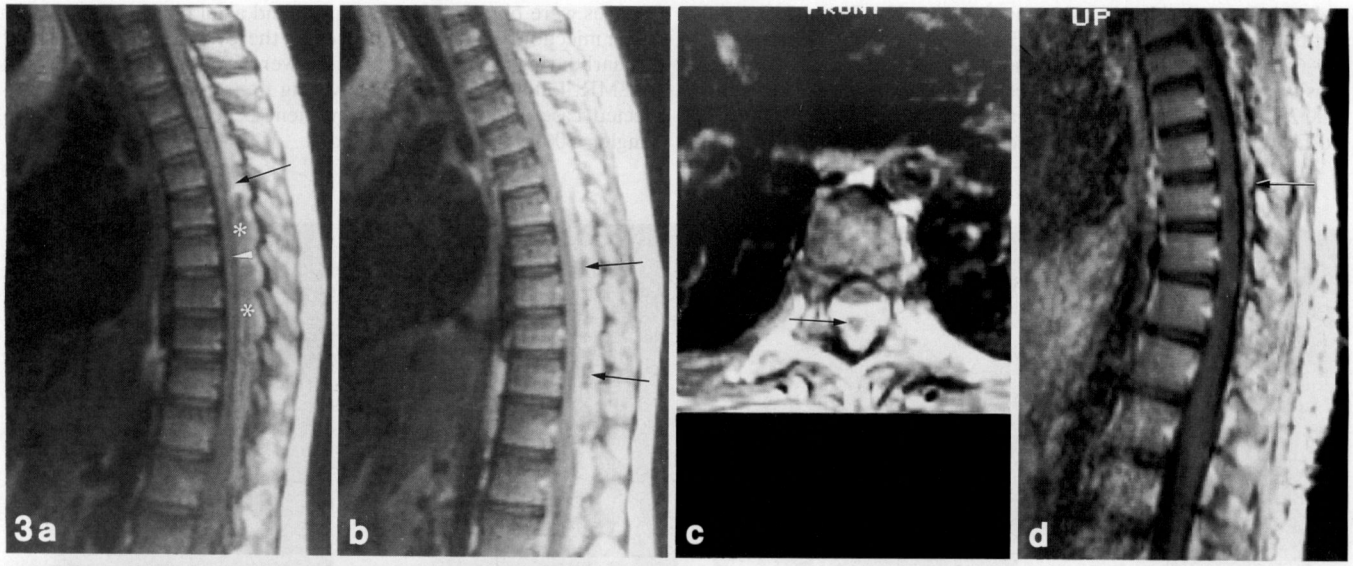


Fig. 1. **a** T1-weighted sagittal image (SE450/20) of the lumbar spine: diffuse abnormal signal intensity in the spinal canal, slightly higher than that of normal CSF, obscuring the subarachnoid space. **b** Gd-enhanced T1-weighted sagittal image shows linear contrast enhancement along the dura mater *(arrowheads)*. The unenhanced portion anterior to it represents a collection of pus. The subarachnoid space is obliterated and the conus medullaris and cauda equina are not identified. Thick band-like enhancement along the posterior margin of the vertebral bodies *(arrows)* may represent epidural venous stasis and/or cellulitis. **c** Follow-up CEMR 14 weeks after the first examination. Dural enhancement is no longer seen and there is only minimal enhancement of the epidural venous plexus, probably a normal finding. The subarachnoid space is clearly seen

Fig. 2. **a** T1-weighted sagittal image with Gd-DTPA. Unenhanced pus in the abscess cavity *(asterisks)* is seen in the posterior epidural space with anterior displacement of the enhancing dura mater *(arrowheads)* from L3 to L5. There is also abnormal contrast enhancement in the dorsal epidural space to the level of T12, suggesting upwards extension of the inflammatory process *(arrow)*. **b,c** Follow-up CEMR one week after the initial scan. Although the patient was stable neurologically, he had persistent fever and leukocytosis. The size of the abscess cavity has decreased in the lower lumbar region, with an increase of the subarachnoid space. However, intense peripheral contrast enhancement in the dorsal epidural space obscures the thoracic subarachnoid space *(arrows)*. **d,e** CEMR 13 weeks after the initial scan. The patient had responded to antibiotic therapy and was neurologically stable. However, abnormal contrast enhancement persists in the area of the surgical scar *(arrows)*. There is no abnormal contrast enhancement in the thoracic epidural space



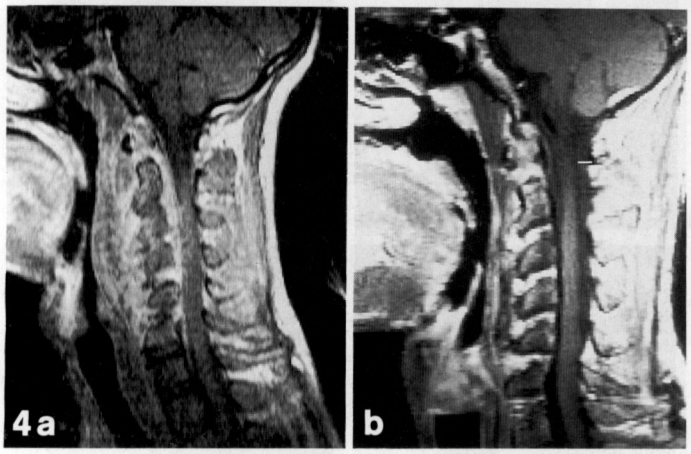


Fig. 3. **a** T1-weighted sagittal image (SE 572/30) shows a dorsal epidural mass of the same intensity as the spinal cord *(asterisks)*. Normal posterior epidural fat *(arrow)* is obliterated. The thoracic cord is displaced anteriorly *(arrowheads)*. Enhanced sagittal (**b**) and axial (**c**) T1-weighted images reveal significant contrast enhancement of an epidural mass with unenhanced areas, presumably pus, within it *(arrows)*. **d** Sagittal CEMR 45 days after the initial examination. The patient was symptom free. There is clear definition of the space thoracic subarachnoid. Minimal enhancement is seen in the posterior epidural area, considered to be a postoperative scar *(arrow)*; it was also seen at six month follow-up

Fig. 4. **a** Gd-enhanced image shows diffuse homogenous contrast enhancement anteriorly in the paraspinal and epidural regions, and in the C2–C6 disks.
b Follow-up CEMR five weeks after completion of antibiotic therapy. The patient had no signs of inflammation and laborary data were normal. Complete resolution of the anterior spinal and epidural masses is seen, but contrast enhancement persists in several disk spaces

Results

Initial images

In two of the four patients with diffuse epidural abscesses, the lesion involved the entire spinal canal, and the subarachnoid space was not seen well on unenhanced T1-weighted images (Fig. 1 a). On T2-weighted images, heterogenous signal intensity was seen in the spinal canal and it was difficult to identify the subarachnoid space. However, CEMRI showed linear contrast enhancement along the dura mater of part or all of the spinal canal, best demonstrated on sagittal images. There was also thick band-like enhancement along the dorsal surface of the vertebral bodies, which looked like engorged epidural veins. Between these enhancing structures, an unenhanced portion was noted in the ventral epidural space (Fig. 1 b); this was thought to represent a collection of pus, suspicion confirmed at surgery.

Another patient had a large, diffuse epidural abscess, with pus mainly in the dorsal aspect of the lower lumbar region. Abnormal contrast enhancement of the dorsal epidural space extended up to the T12 level (Fig. 2 a).

The last of the diffuse abscesses was in the posterior thoracic spinal canal, and was clearly demarcated by the lack of epidural fat on unenhanced T1-weighted sagittal images (Fig. 3 a). CEMR demonstrated thick, irregular contrast enhancement in the dorsal epidural space (Fig. 3 b,c) thought to represent abundant granulation tissue mixed with pus, as proved surgically.

The primary or concomitant focus of infection was found in three of the four patients with diffuse epidural abscess: a retroperitoneal abscess, osteomyelitis/diskitis of the lumbar vertebral bodies and facet arthritis of the lower lumbar spine.

NCMR showed focal epidural abscesses in three of four patients. They were isointense with the spinal cord on T1-weighted images, and of higher intensity on T2 weighting. CEMR showed diffuse homogeneous contrast enhancement, predominantly ventral to the dural sac (Fig. 4). One patient did not show an epidural soft tissue mass on NCMR or sagittal CEMR, but axial CEMR revealed circumferiential contrast enhancement in the epidural space.

Osteomyelitis or diskitis was identified adjacent to all the focal epidural abscesses, with diffuse contrast enhancement of the vertebral bodies or disk spaces.

Follow-up studies

Three of four patient with diffuse abscesses responded to initial treatment by drainage and antibiotics. One of the three underwent drainage of a retroperitoneal abscess but the epidural abscess was treated medically; a follow-up MRI showed a decrease in the size of the epidural pus collection, and the subarachnoid space became visible within five days of the initiation of therapy. However, contrast enhancement along the dura mater and the dorsal surface of the vertebral bodies persisted, having almost disappeared only at 14th week follow-up CEMR (Fig. 1c). The other two patients underwent surgical drainage, and follow-up MRI demonstrated significant decrease or disappearance of their abscesses and clear definition of the subarachnoid space. Persistent moderate contrast enhancement in the posterior epidural area at the site of surgical drainage was thought to represent postsurgical scar (Figs. 2d, 3d). In one patient, MRI showed extension of the abscess into the thoracic spine one week after surgical drainage, accompanied by leukocytosis and fever (Fig. 2b,c). Aspiration of the abscess revealed concomitant anaerobic infection. A change of the antibiotic regime was followed by decrease in size of the abscess and clinical improvement.

One of the four patients with focal abscesses responded well to antibiotic therapy and follow-up MRI showed a significant decrease in the size of a paraspinal mass and the epidural abscess. However, residual contrast enhancement in the cervical intervertebral disks at the end of the 6 week course of antibiotic therapy persisted for an additional 5 weeks, despite a lack of symptoms and normal laboratory data (Fig. 4b). The other three patients did not improve clinically or radiologically, despite medical and/or surgical treatment.

Discussion

There have been several reports on MRI in epidural abscess. Angtuaco et al. [7] reported that MRI clearly showed epidural abscesses when radionuclide bone scans and labeled leukocyte scans were negative. In their analysis of 21 patients with epidural abscesses, Post et al. [6] reported that NCMR showed focal epidural abscesses extending from osteomyelitis as clearly demarcated masses, obviating myelography. However, they found diffuse abscesses without osteomyelitis difficult to see and thought myelography with high-resolution CT is superior to MRI in such cases. They speculated that this was because the signal characteristics of infected cerebrospinal fluid (CSF) can not be differentiated from those of epidural abscesses. Masaryk and Modic [12] also mentioned that collections of inflammatory fluid may be difficult to differentiate from the CSF. However, as shown here, the difficulty in diagnosing diffuse epidural abscess by NCMR was probably due mainly to obliteration of the normal CSF-containing surbarachnoid space by the epidural lesion.

The advantages of Gd-DTPA in the diagnosis of inflammatory disease of the central nervous system have been established [13–15]. Post et al. [9] recently reviewed 21 patients with epidural abscess diagnosed by CEMR and found that the majority of the abscesses enhanced densely and homogenously [9]. They also pointed out the potential limitations of CEMR for diagnosing abscesses which contain large quantities of fresh pus but have little surrounding granulation tissue.

The findings in our cases of diffuse abscess have not been fully described previously. First, the linear contrast enhancement along the dura mater in three of the four patients may represent inflammation of the dura mater itself or peridural inflammatory reaction. This enhancement surrounded unenhanced regions suggestive of pus in the epidural space and facilitated differentiating them from intradural lesions. Second, when of diffuse epidural abscesses originate in the lumbar region, abscesses in the cervical and upper thoracic regions may not exhibit significant discrete contrast enhancement; but are accompanied by disappearance of the normal subarachnoid space, mimicking an intradural lesion. This is probably due to rapid extension of liquid pus with minimal inflammatory reaction around it. In these cases, it may be necessary to image the entire spine.

The exact cause of band-like contrast enhancement along the posterior margin of the vertebral bodies and ventral to the pus collections seen in two of our patients with a diffuse epidural abscess is not clear. It may represent vascular stasis in the epidural venous plexus due to an obstructive process in the spinal canal associated with inflammation. Cellulitis or granulation around the pus may also be responsible.

Thick, irregular contrast enhancement corresponding to the amount of granulation tissue and pus formation in the abscess was also seen in one of our patients, and in the series of Post et al. [9].

Focal epidural abscesses showed as a localized epidural lesion adjacent to the osteomyelitis and diskitis, as described in previous reports [6, 7, 9]. Homogenous contrast enhancement without appreciable unenhanced areas in our patients is probably due to abundant granulation tissue or early cellulitis with little pus formation.

Serial CEMR is valuable for assessing changes in size of the abscess and the degree of thecal compression. In patients who do not respond to therapy, CEMR is useful for determining the cause and the treatment of choice. Patients with poor neurological recovery from and persistent abscess cavities may be candidates for second operations or for revision of antibiotic therapy.

Enhancement of concomitant foci of infection adjacent to the abscess can persist despite disappearance of the abscess. It probably represents chronic sterile granulomatous change, as in brain abscesses [16, 17]. Residual contrast enhancement itself does not necessitate continued antibiotic therapy. However, follow-up MRI and close medical observation are recommended for one to two weeks after treatment has been completed, particularly if the patient has predisposing factors such as diabetes mellitus or is immunosuppressed.

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