Spinal Epidural Abscess: Evaluation with Gadolinium-enhanced MR Imaging[1]

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Magnetic resonance (MR) imaging with gadopentetate dimeglumine was performed in 25 patients with spinal epidural abscess (SEA). Seventeen of 25 patients underwent follow-up MR imaging. The studies were retrospectively reviewed. In 20 patients, diskitis was the primary infectious foci; however, five patients developed diffuse SEA without diskitis. The two most common MR appearances were (*a*) homogeneous or heterogeneous enhancement of the solid portion of the SEA and (*b*) thin or thick enhancement around the liquefied collections of pus. Dural enhancement was frequently seen in patients with lengthy vertebral involvement of SEA. Engorgement of the epidural venous plexus or basivertebral veins was occasionally observed. The changes in abscess size noted on follow-up studies correlated well with clinical improvement or deterioration in most patients. Persistent contrast enhancement, however, was frequently noted at the site of diskitis, osteomyelitis, or surgical drainage sites despite clinical improvement. Careful correlation of MR imaging findings with clinical findings and laboratory data is important in predicting prognosis for these patients.

Abbreviation: SEA = spinal epidural abscess

Index terms: Spinal cord, compression, 30.38 • Spinal cord, infection, 30.242, 30.25 • Spondylitis, 30.242, 30.25

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See the commentary by Quencer following this article.

■ INTRODUCTION

Spinal epidural abscess (SEA) can cause marked neurologic morbidity. Its prevalence is apparently increasing (1,2), and this trend has been confirmed during the past several years at our institution. SEA can occur in patients with chronic illnesses such as diabetes mellitus, in those with histories of intravenous drug abuse, and in those with underlying immunodeficiency (1,2). Prompt diagnosis and treatment are important because SEA may lead to paraplegia, quadriplegia, and death if untreated or if treatment is inadequate or delayed.

Although myelography and computed tomography (CT) have previously been used to diagnose spinal infection and SEA, magnetic resonance (MR) imaging has recently emerged as the modality of choice for imaging of these entities (3–10). The importance of using gadopentetate dimeglumine for diagnosing SEA has been underscored (6,9,10).

This article describes the spectra of MR imaging findings in SEA and correlation between results from serial follow-up MR imaging with gadopentetate dimeglumine and the clinical manifestations of SEA.

■ MATERIALS AND METHODS

The images from MR examinations of 25 patients with SEA were retrospectively evaluated.

MR imaging was performed using a 1.5-T apparatus (Siemens Medical Systems, Iselin, NJ). Studies performed before the administration of contrast material included sagittal and axial T1-weighted spin-echo images with repetition times of 450–650 msec, echo times of 17–20 msec, and two signals averaged, as well as proton-density and T2-weighted sagittal spin-echo images with repetition times of 2,000–3,500 msec, echo times of 25–90 msec, and one signal. Postcontrast sagittal and axial T1-weighted images were obtained after the intravenous administration of 0.1 mmol/kg of gadopentetate dimeglumine (Magnevist; Berlex Laboratories, Wayne, NJ). If extension of the SEA was thought to be above or below the site examined, additional postcontrast images were obtained in the sagittal plane and in the axial plane at selected levels.

Eight patients underwent only one contrast material–enhanced MR imaging examination, and follow-up examinations were not performed on these patients for various reasons. Seventeen patients underwent follow-up contrast-enhanced MR imaging examinations at various intervals after their initial studies. Among them, 12 patients underwent early follow-up studies (within the first 3 weeks), and 13 patients underwent late follow-up studies (from 3 weeks to 260 days after the initial studies).

Table 1
Predisposing Factors for the Development of SEA

Factor	No. of Patients*
Diabetes mellitus	10
Intravenous drug abuse	8
Trauma	4
Hemodialysis	1
Dental surgery	2
Epidural catheter	1
Unknown	3

* Thirteen patients each had multiple factors.

■ RESULTS

• Clinical Findings

The series consisted of 14 male and 11 female patients whose ages ranged from 8 months to 79 years (average, 50 years). Patients with diabetes mellitus and known intravenous drug abuse were most commonly affected (Table 1). *Staphylococcus aureus* was by far the most frequent organism causing SEA.

SEA was encountered in all segments of the spinal canal and column (Fig 1). In eight patients, the SEA involved six or more vertebral segments, and these patients were considered to have diffuse SEA. In three of the eight patients, the entire spinal canal was involved. Focal spinal canal involvement and involvement of five or fewer vertebral segments were observed in 17 patients, and they were classified as having focal SEA. In one patient, focal SEA was observed as separate lesions in the thoracic and lumbar regions.

Concomitant diskitis or osteomyelitis adjacent to the SEA was a common occurrence, especially in patients with focal SEA (Table 2). However, only three of eight patients with diffuse SEA had associated diskitis or osteomyelitis, and four of the eight patients had facet infection or abscess in the paraspinal and retroperitoneal region as a likely source of SEA.

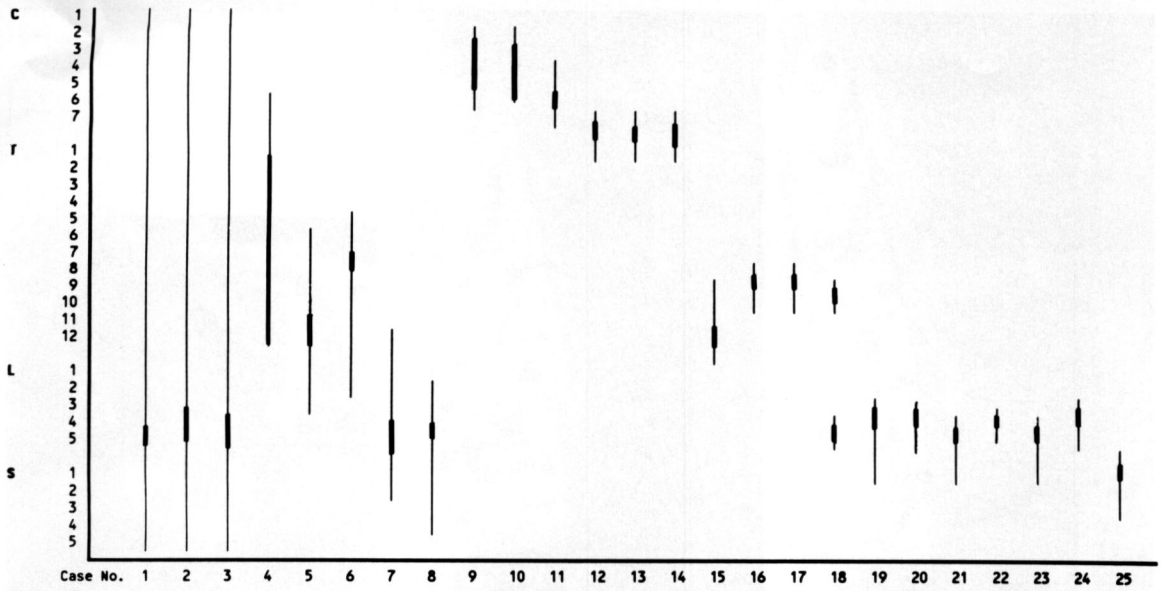


Figure 1. Graph illustrates the caudocranial extent of SEA in relation to the vertebral bodies. The thicker portion of the line indicates the site where infection started.

Table 2
Concomitant Infections in Patients with SEA

Infection	Patients with Diffuse SEA (n = 8)*	Patients with Focal SEA (n = 17)
Diskitis or osteomyelitis	3	17
Facet infection	1	0
Posterior paraspinal abscess	2	2
Retroperitoneal abscess	1	1

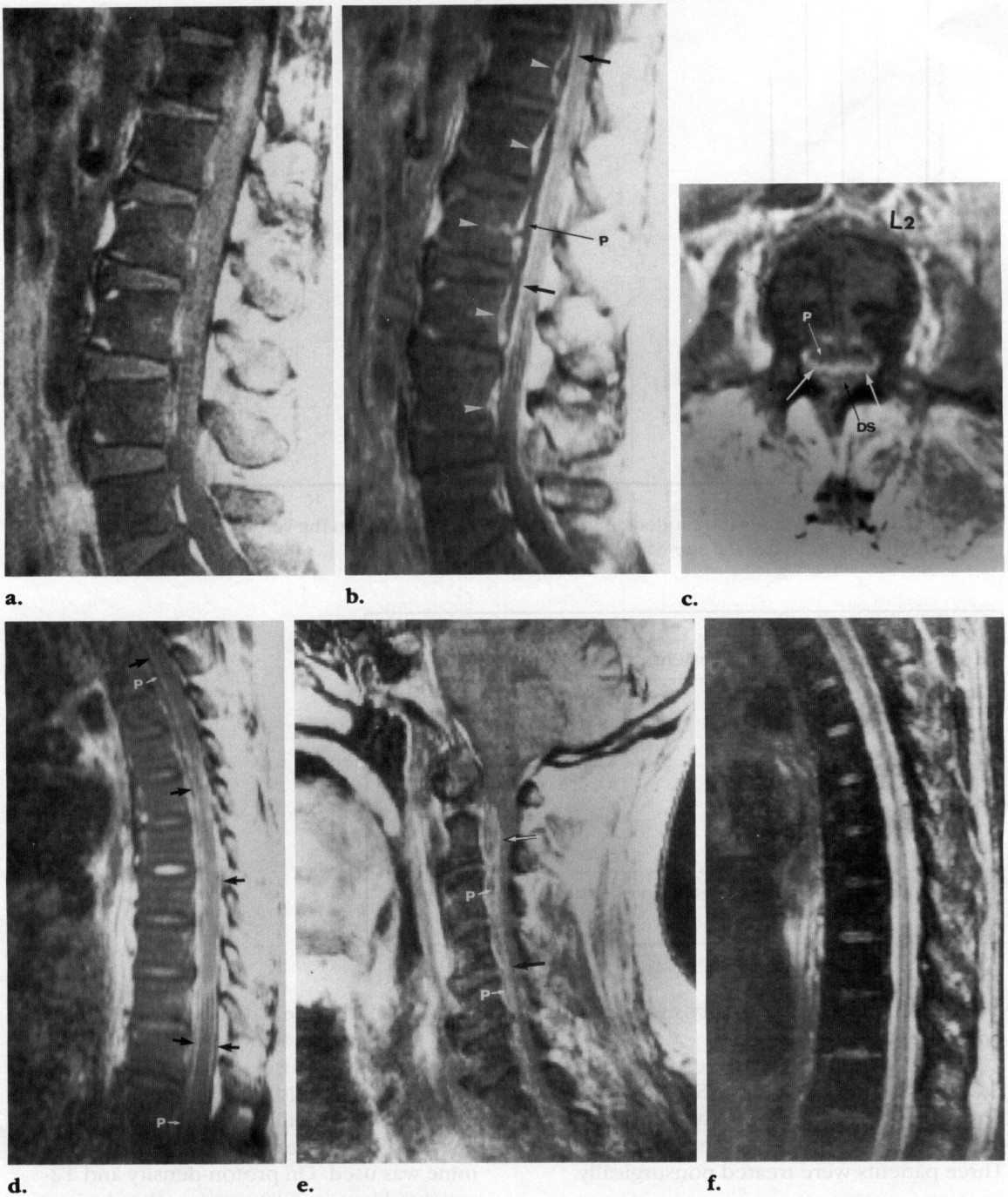
* No concomitant infection present in one patient with a diffuse SEA.

No concomitant infectious focus was found in one patient with diffuse SEA.

Twenty patients underwent surgical decompression and drainage of the SEA. Open biopsy procedures were performed in two patients to identify the causative organisms. Three patients were treated nonsurgically.

• Initial MR Imaging

Precontrast Studies.—SEA appeared isointense or of slightly high signal intensity compared with that of the spinal cord on T1-weighted images. Low signal intensity representing a liquid collection of pus was also observed in 10 patients. The normal dorsal epidural fat was obliterated in four patients. In cases of diffuse SEA, the signal intensity of cerebrospinal fluid in the subarachnoid space was not visualized on T1-weighted images at the involved segment due to total encasement and displacement of the dural sac (Figs 2, 3). In three patients, cerebrospinal fluid could not be seen throughout the entire spinal canal. Because of this nonvisualization of the subarachnoid space, these lesions were often difficult to differentiate from intradural abnormalities unless gadopentetate dimeglumine was used. On proton-density and T2-weighted images, SEA was visualized as an area of high signal intensity and was therefore difficult to differentiate from cerebrospinal fluid (Fig 2). However, proton-density and T2-weighted images helped demonstrate the areas of diskitis and osteomyelitis and paravertebral abscess as high-signal-intensity lesions. Diffusely diminished signal intensity from the marrow of the vertebral bodies was noted in 17 patients on T1-weighted images (Figs 2, 3).



a. b. c.

d. e. f.

Figure 2. Case 1. A 51-year-old man with diabetes mellitus developed a retroperitoneal abscess 3 weeks after dental surgery. The abscess was surgically drained. He later complained of low back pain and fever. **(a)** Precontrast sagittal T1-weighted image exhibits diffuse abnormal signal intensity slightly higher than that of normal cerebrospinal fluid in the spinal canal, obscuring the entire subarachnoid space in the lumbar and lower thoracic regions. Diffusely diminished marrow signal intensity is noted in all vertebral bodies. **(b)** Postcontrast sagittal T1-weighted image shows linear enhancement along the dura mater (arrows). The unenhancing region anterior to the dura mater represents a collection of pus (*P*). The subarachnoid space is totally compressed by the epidural mass. Thick bandlike enhancement along posterior margins of the vertebral bodies may represent epidural venous stasis (arrowheads). **(c)** On an axial postcontrast image obtained at the L-2 level, the dural sac (*DS*) is compressed posteriorly by pus (*P*), which is surrounded by linear enhancement (arrows). **(d, e)** On sagittal images, contrast enhancement along the dura mater extends superiorly to the thoracic and cervical spinal canal (arrows). The subarachnoid space is completely effaced by the unenhanced anterior epidural collection of pus (*P*). **(f)** On the T2-weighted image of the thoracic region, the SEA is indistinguishable from cerebrospinal fluid (*Fig 2 continues*).

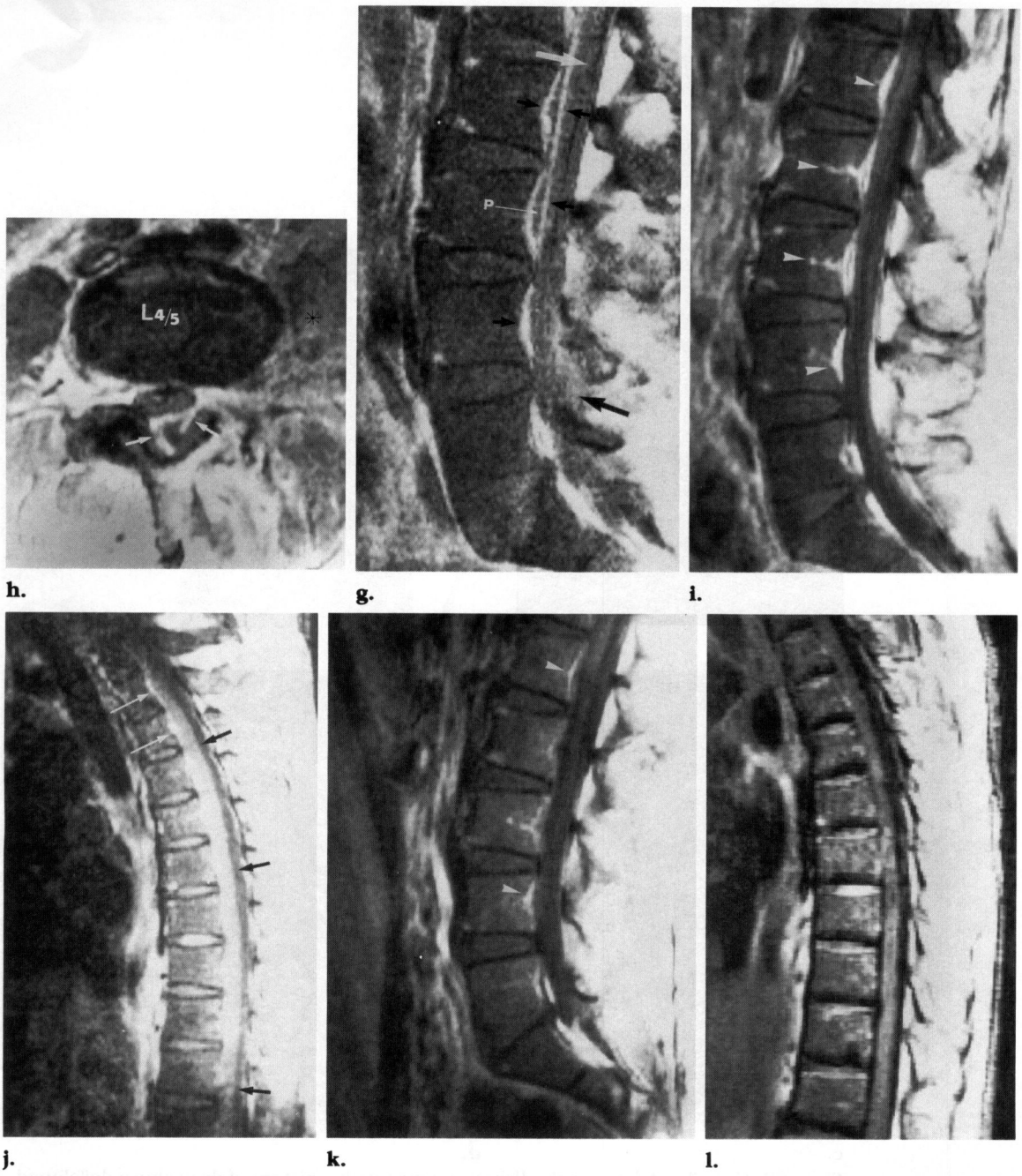


Figure 2. Case 1 (*continued*). **(g, h)** Follow-up postcontrast T1-weighted images were obtained 5 days after initiation of antibiotic therapy. **(g)** On the sagittal section, the collection of pus (*P*) in the anterior epidural space has decreased in size, and the normal cerebrospinal fluid signal intensity in the subarachnoid space (white arrow) is now better visualized due to decreased epidural mass effect. Enhancement along the dura mater and vertebral bodies is still present (short black arrows). At the L4-5 level, there is a low-signal-intensity mass in the dorsal epidural space obliterating normal epidural fat (long black arrow). **(h)** On the axial section, unenhancing pus in the dorsal epidural space is surrounded by linear enhancement (arrows). Note the minimal enhancement of the left psoas, indicating residual inflammation due to the retroperitoneal abscess (*). **(i, j)** Postcontrast sagittal T1-weighted images of the lumbar and thoracic regions obtained 2 weeks later demonstrate marked improvement in visualization of the subarachnoid space, particularly in the lumbar region. The subarachnoid space posterior to the thoracic cord is now visualized (black arrows in **j**), but cerebrospinal fluid anterior to the cord is not seen, suggesting residual anterior epidural abscess. The anterior and posterior SEA has nearly resolved in the lumbar region. Minimal linear enhancement is noted in the upper thoracic epidural area (white arrows in **j**). Prominent enhancement along vertebral bodies and basivertebral veins persists in the lumbar region (arrowheads in **i**). **(k, l)** On postcontrast sagittal T1-weighted images obtained 3 months later, the subarachnoid space of the lumbar and thoracic regions is clearly observed, and only minimal enhancement is noted in the basivertebral veins (arrowheads in **k**). The patient was symptom free and ambulatory without assistance.

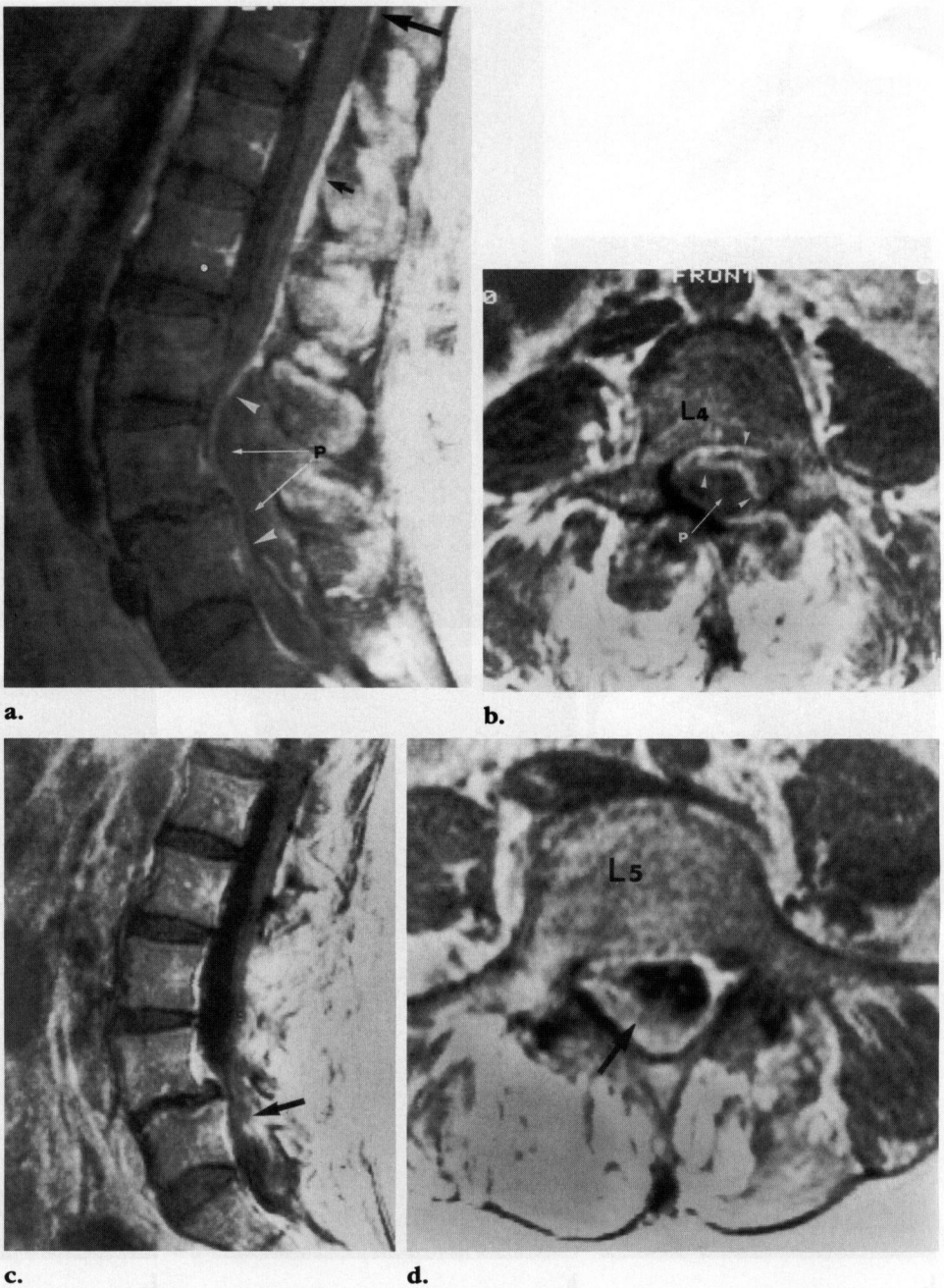
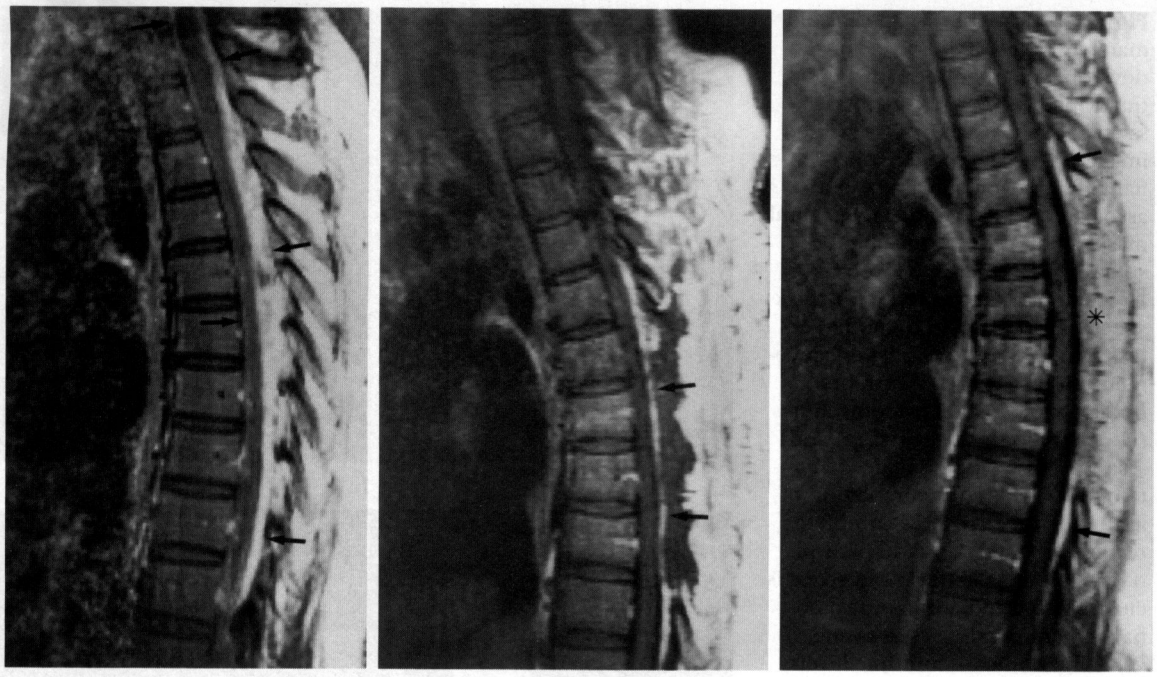
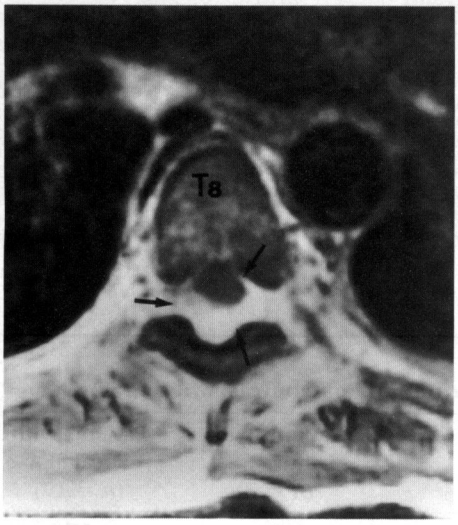


Figure 3. Case 7. A 59-year-old diabetic woman with a history of drug abuse developed progressive paraparesis, back pain, and fever. **(a, b)** Postcontrast sagittal **(a)** and axial **(b)** T1-weighted images demonstrate a nonenhancing posterior epidural mass of low signal intensity in the lower lumbar and sacral regions, indicating the presence of a collection of pus (*P*). The enhancing dural sac is displaced anteriorly (arrowheads), and the subarachnoid space in these regions is markedly compromised. The dural enhancement extends upward to the T-11 level (large arrow in **a**). High-signal-intensity band in the upper lumbar spine represents normal dorsal epidural fat (small arrow in **a**). There is retrolisthesis of the L-5 vertebral body associated with disk narrowing at L4-5, probably due to a degenerative process. **(c, d)** Postcontrast sagittal **(c)** and axial **(d)** T1-weighted images obtained 5 months after the patient underwent abscess drainage show contrast enhancement in the scar tissue around the dural sac (arrow). The patient was ambulatory with a walker and essentially symptom free except for mild low back pain. Her erythrocyte sedimentation rate was normal.



a. c. d.

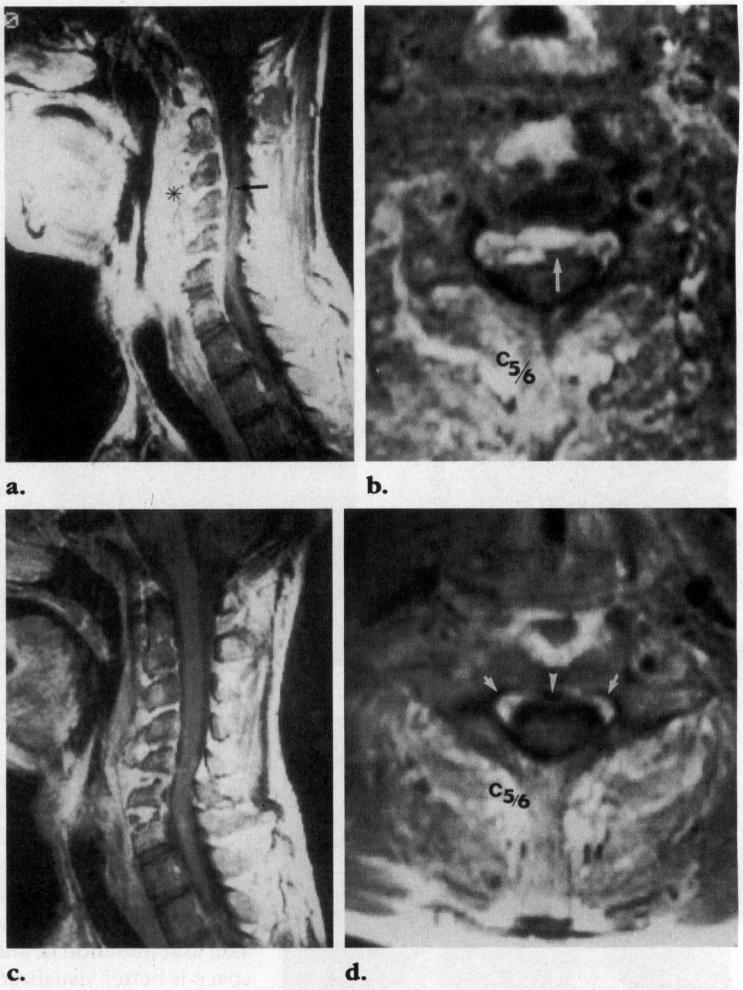


b.

Figure 4. Case 4. A 37-year-old woman with a history of intravenous drug abuse complained of back pain. **(a, b)** Postcontrast sagittal **(a)** and axial **(b)** T1-weighted images show an SEA throughout the entire thoracic spine, especially involving the posterior epidural space (arrows). Note the complete obliteration of the subarachnoid space by a circumferential epidural abscess. **(c)** On the postcontrast sagittal T1-weighted image obtained 1 week after surgical decompression, abscess drainage, and initiation of antibiotic therapy, the subarachnoid space is better visualized. Linear dural enhancement is noted only at the surgical site (arrows). **(d)** On the postcontrast image obtained 2 months later, the subarachnoid space is clearly visualized. There is diffuse enhancement in the postoperative scar tissue (*). High-signal-intensity bands above and below the scar tissue represent normal epidural fat (arrows). The patient was symptom free with a normal erythrocyte sedimentation rate.

Postcontrast Studies.—The most common finding in both diffuse and focal SEA cases was diffuse enhancement at the site of the solid component of the SEA, in either a homogeneous or heterogeneous fashion (Figs 4–7); this was observed in 18 patients (72%) (Table 3). The second most common finding was thin or thick enhancement around a liquefied abscess, that is, a collection of pus that appeared to be of low signal intensity surrounded by granulation tissue (Figs 2, 7, 8). This was noted in 10 of 25 patients (40%) but was more frequently observed in patients with diffuse SEA (six of eight [75%]).

Figure 5. Case 10. A 35-year-old man with a history of drug abuse developed acute neck pain and quadriplegia. **(a, b)** Postcontrast sagittal **(a)** and axial **(b)** T1-weighted images show a homogeneously enhancing paraspinal mass (* in **a**) associated with an anterior epidural mass (arrow) compromising the spinal cord at the C5-6 level. Enhancement is also noted in the disk spaces. There is mild to moderate loss of height and contrast enhancement in the C-3 to C-7 vertebral bodies, indicative of osteomyelitis. **(c, d)** Postcontrast sagittal **(c)** and axial **(d)** T1-weighted images obtained 5 weeks after completion of antibiotic therapy show complete resolution of the SEA and paraspinal mass. The subarachnoid space is clearly visualized, but contrast enhancement persists in disks and vertebral bodies at multiple levels, presumably representing sterile granulation tissue. Normal enhancement of the epidural vein is seen on the axial view (arrows in **d**). Note symmetric enhancement of the veins, with sparing of the portion of the posterior longitudinal ligament (arrowhead in **d**). The patient had no clinical signs of inflammation, and his erythrocyte sedimentation rate was normal.



a. **b.** **c.** **d.**

Table 3
Summary of Findings at Initial Postcontrast MR Imaging

Finding	Patients with Diffuse SEA (n = 8)	Patients with Focal SEA (n = 17)
Disk or bone enhancement	3	17
Diffuse enhancement	5	13
Peripheral enhancement around pus	6	4
Linear dural enhancement on sagittal view	6	0
Epidural venous engorgement	3	6

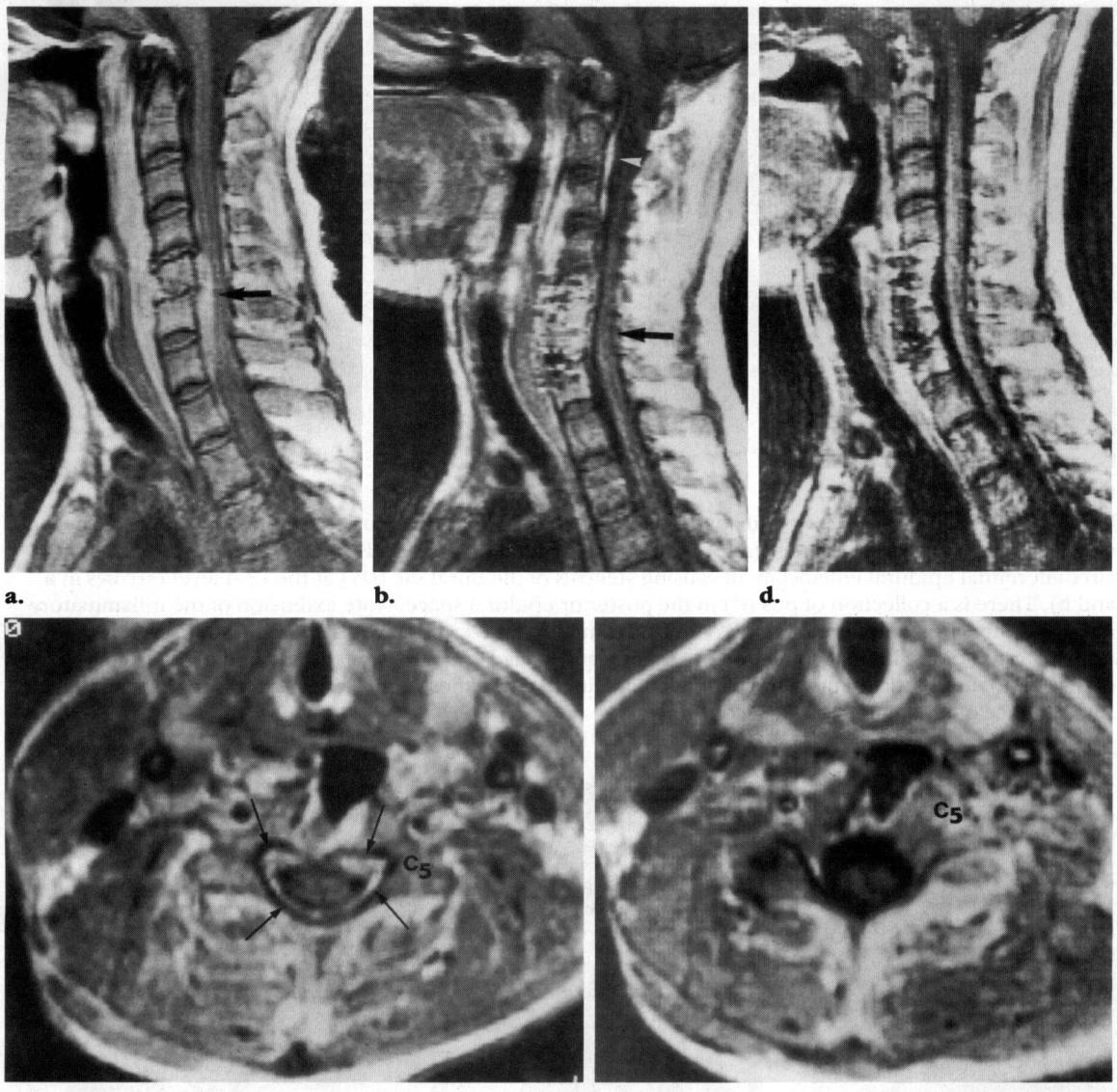
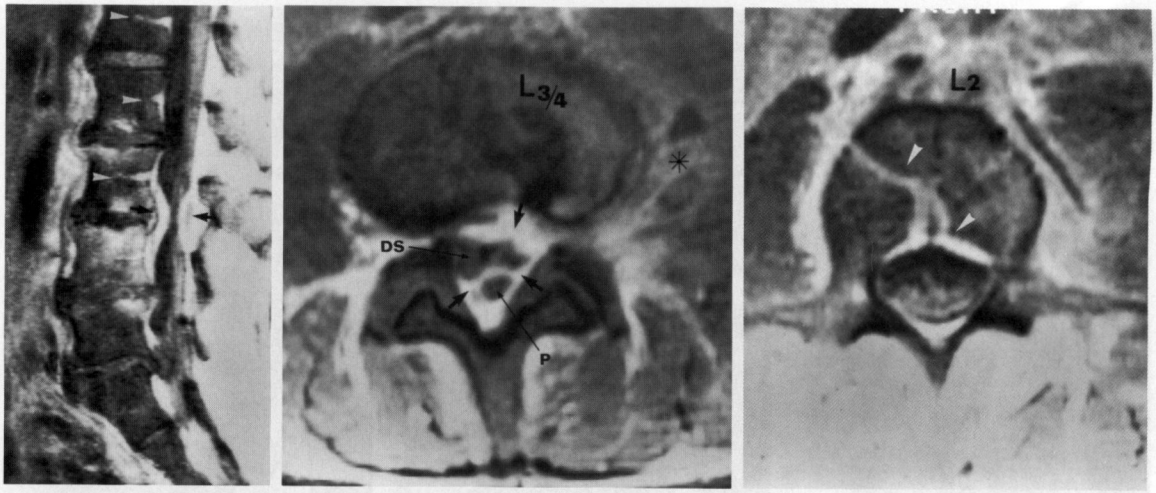
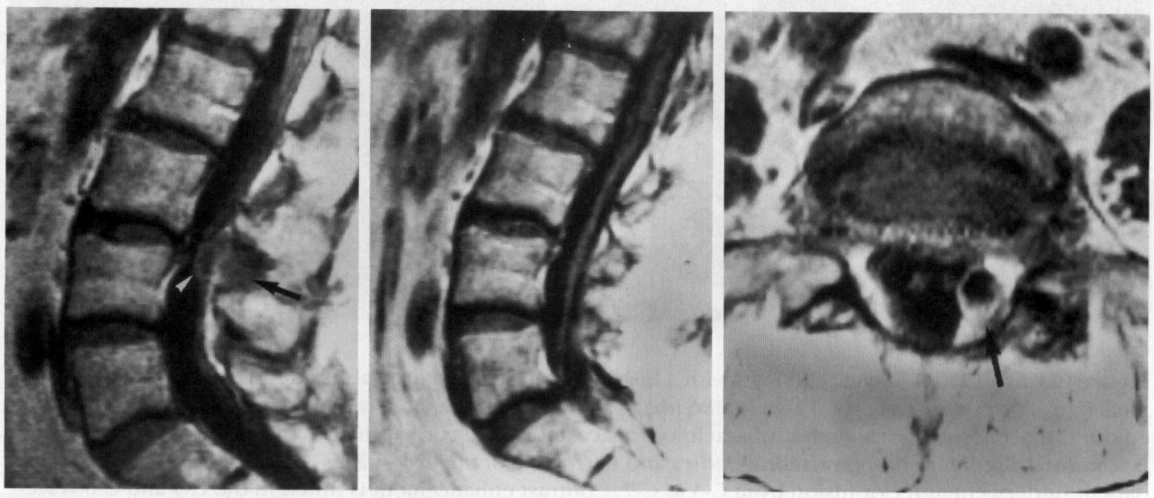


Figure 6. Case 11. A 38-year-old man with a history of intravenous drug abuse developed fever and paraplegia. **(a)** Postcontrast sagittal T1-weighted image shows a homogeneously enhancing mass in the anterior epidural space, compromising the subarachnoid space and spinal cord at the C-4 to T-1 levels (arrow). There is abnormal alignment of the vertebral bodies and narrowing of the disk spaces at the C-4 to C-6 levels. The patient underwent anterior fusion from C-5 to C-7. Contrast enhancement is noted in the C-5 and C-6 vertebral bodies, indicative of osteomyelitis. **(b, c)** Postcontrast sagittal **(b)** and axial **(c)** T1-weighted images obtained 2 weeks after surgery demonstrate enhancement of the vertebral bodies at the surgical site. Moderate compression of the spinal cord is still observed (arrow in **b**), and there is engorgement of the anterior epidural veins at the C-2 to C-3 level (arrowhead in **b**). Circumferential epidural enhancement compromising the subarachnoid space is seen on the axial view (arrows in **c**). **(d, e)** Postcontrast sagittal **(d)** and axial **(e)** T1-weighted images obtained 4 weeks later show persistent contrast enhancement of the vertebral bodies at the surgical site, but the epidural space is free of abnormal contrast enhancement. The subarachnoid space is clearly visualized. The previously noted engorged epidural veins at the C-2 to C-3 level are no longer visible. The patient was asymptomatic.



a. b. c.

Figure 7. Case 20. A 48-year-old diabetic man with a history of drug abuse developed fever, backache, and weakness of his lower extremities. **(a–c)** Postcontrast sagittal **(a)** and axial **(b, c)** T1-weighted images show circumferential epidural enhancement causing stenosis of the dural sac (*DS*) at the L3-4 level (arrows in **a** and **b**). There is a collection of pus (*P*) in the posterior epidural space. Note extension of the inflammatory mass into the left paravertebral region (* in **b**). Prominent basivertebral veins are seen in the region of the upper portion of the lumbar spine on both sagittal and axial views (arrowheads in **a** and **c**). After completion of 8 weeks of antibiotic therapy, the patient complained of weakness of his lower extremities but had no clinical evidence of inflammation (*Fig 7 continues*).



a. b. c.

Figure 8. Case 22. A 70-year-old woman complained of fever and low back pain that developed 1 week after she received a steroid injection into her facet joints. **(a)** Postcontrast sagittal T1-weighted image shows a dorsal epidural mass at the L3-4 level (arrow) with minimal peripheral enhancement (arrowhead). The posterior epidural fat is obliterated, and the dural sac is markedly compressed. Pus was drained at surgery. The patient had an uneventful postoperative course but again became febrile after a 4-week course of antibiotic therapy was completed. **(b)** Postcontrast sagittal T1-weighted image shows no definite abnormality. The posterior epidural mass is no longer visible at the L3-4 level. **(c)** Axial T1-weighted image obtained at the L-5 to S-1 level, however, demonstrates a localized collection of pus surrounded by linear contrast enhancement in the posterior epidural space (arrow). The pus was drained, and the patient began an additional 4-week course of antibiotic therapy.

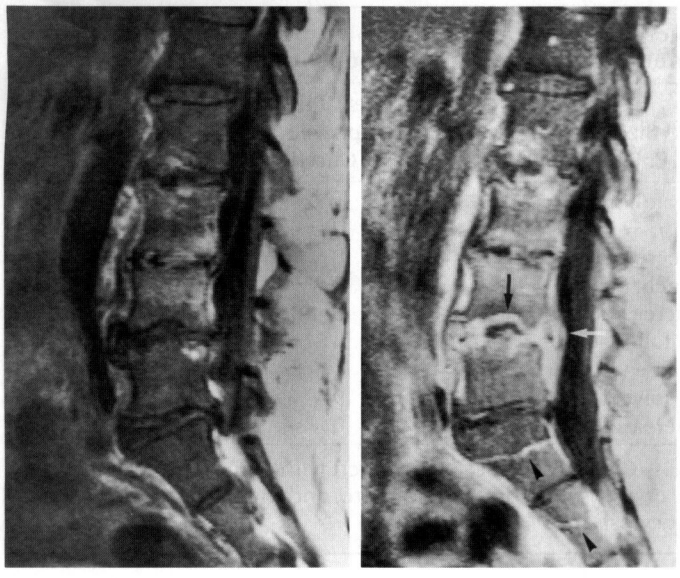
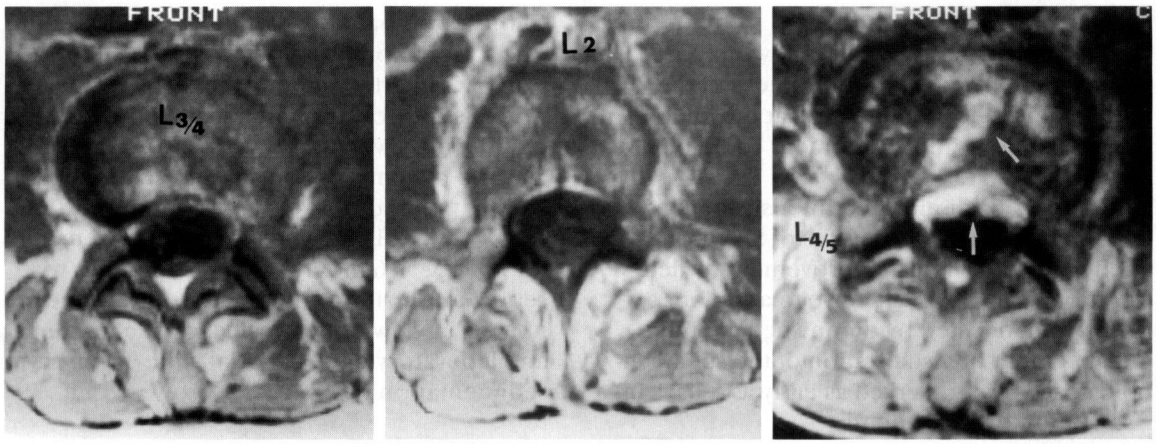


Figure 7. Case 20 *(continued).* **(d–f)** On follow-up postcontrast sagittal **(d)** and axial **(e, f)** T1-weighted images, abnormal enhancement in the epidural and paravertebral spaces has nearly resolved. Dural sac compression and prominence of the basivertebral veins are no longer seen. There is slight enhancement of the L-3 and L-4 vertebral bodies. Five months later, the patient again developed fever and low back pain. **(g, h)** Postcontrast sagittal **(g)** and axial **(h)** T1-weighted images demonstrate a new lesion at L4-5. There is diskitis with an associated epidural abscess mainly in the anterior epidural space (arrows). Prominent epidural veins are seen at S-1 and S-2 (arrowheads in **g**). The patient is currently receiving continuous antibiotic therapy.

d. **g.**



e. **f.** **h.**

Linear enhancement along the compressed dura mater was observed in six of eight (75%) patients with diffuse SEA on sagittal images (Figs 2, 3). This finding was not observed in any patient with focal SEA. In three patients with diffuse SEA, this linear enhancement was observed throughout the entire spinal canal on the sagittal images, and it facilitated correct assessment of the extent of the abscess (Fig 2).

Linear enhancement along the posterior margin of the vertebral column above or below the main sites of SEA was noted in nine of 25 patients (36%). This was seen on sagittal images and was thought to represent prominent anterior epidural veins or the basivertebral venous plexus (Figs 2, 6, 7). This finding was observed in diffuse and focal SEA cases with similar frequency (38% vs 35%, respectively).

Sagittal views were the most useful projections for assessment of cephalic or caudal extensions of SEA. However, axial projections were also mandatory, not only for confirming the diagnosis of SEA, but also for defining the exact sites of cellulitis or granulation tissues and collections of pus in relation to the dural sac or bony structures. Axial views were superior to sagittal images for demonstrating concomitant abscess formation in the paravertebral region. When cephalic or caudal extensions of infection from the primary foci were suspected, axial sections obtained at selected levels often helped confirm this manifestation by demonstrating abnormal epidural contrast enhancement.

Table 4
Summary of Findings at Follow-up Postcontrast MR Imaging

SEA Size or Contrast Enhancement	Patients Clinically Improved or Cured	Patients Clinically Unchanged or Worse
Early follow-up (n = 12)		
Decreased	8	0
Unchanged	1	0
Increased	0	3
Late follow-up (n = 13)		
Decreased or resolved	9	2
Increased	0	2

Note.—Clinical status as determined with neurologic examination, erythrocyte sedimentation rate, and the presence of fever or leukocytosis.

• Follow-up MR Imaging

Twelve patients underwent follow-up postcontrast MR imaging within 3 weeks of the initial MR imaging studies so that the effectiveness of their initial treatment could be assessed.

Increased or diminished intensity of contrast enhancement at the sites of SEA correlated well with clinical improvement or deterioration (Table 4). Response to antibiotic therapy, manifested by diminished contrast enhancement or visualization of the subarachnoid space, was observed as early as 5 days in a small number of patients.

In three patients, an increase in contrast enhancement at the primary foci of infection or at cephalic or caudal extensions of abscess was observed on follow-up MR images. Alterations of treatment regimens such as repeat drainage or administration of a different antibiotic were pursued for those patients.

Follow-up postcontrast MR imaging was performed later than 3 weeks after their initial studies for 13 patients. Decreased, resolved, or increased degrees of contrast enhancement correlated well with clinical improvement or deterioration in all but two patients (Table 4). In these two patients, symptoms worsened due to cord compression caused by subluxation of the vertebral bodies.

Persistent contrast enhancement was frequently observed at the sites of surgical drainage and decompression surgery and at the sites of previous diskitis and osteomyelitis (Figs 3–6). This enhancement was observed for as long as 6 months and was thought to represent sterile granulomatous tissue and fibrosis in view of the normal clinical findings and laboratory data, especially the erythrocyte sedimentation rate.

■ DISCUSSION

Myelography or CT myelography can be effective in the diagnosis of extradural compression or blocking due to SEA. However, these examinations entail the risk of seeding infection into the subarachnoid space, and MR imaging is now regarded as the diagnostic test of choice for detecting SEA (3–10). Precontrast T1-weighted imaging is important for detecting the sites of diskitis or osteomyelitis, which are especially well visualized as focal areas of low signal intensity in the vertebral bodies. If only postcontrast MR imaging is performed, regions of diskitis or osteomyelitis may be homogeneously enhanced and the lesions may be easily overlooked. Obliteration of normal epidural fat by abscesses can also be easily

detected on T1-weighted images. Diffusely decreased signal intensity in the marrow of the vertebral bodies can be visualized on T1-weighted images in many patients with SEA. This finding can also be seen in patients with chronic anemia, acquired immunodeficiency syndrome, diabetes mellitus, or renal failure. Iron deposition in the bone marrow has been suggested as a factor responsible for this finding (11).

Precontrast proton-density and T2-weighted images often fail to demonstrate an abscess because both cerebrospinal fluid and an abscess have high signal intensity (9). It is our opinion, however, that MR imaging with long repetition times is necessary at an initial study because it often shows well the increased signal-intensity areas of osteomyelitis and diskitis, as well as paravertebral abscess or collections of pus associated with SEA.

Postcontrast MR imaging invariably delineates the extent of an abscess and the degree of compression of the thecal sac. It can also be used to help localize a site of potential biopsy (10). Two basic patterns of enhancement of SEA have been described (6,9). The first pattern is homogeneous or heterogeneous enhancement, which represents cellulitis or granulomatous tissues with imbedded microabscesses, indicative of the phlegmonous stage of infection. The second pattern is of an unenhancing central region representing a collection of liquid pus surrounded by thin or thick rim enhancement. These findings were also the predominant features in the present study.

There are two additional important patterns of contrast enhancement: linear enhancement along the dura mater and engorgement of the epidural or basivertebral veins. These enhancement patterns are best visualized in the sagittal planes. Linear dural enhancement probably represents extension of inflammation into the dura mater and may be mistaken for intradural or perimedullary infection, especially when the entire subarachnoid space is compressed by the SEA. This error can be avoided by careful correlation of the findings from sagittal and axial sections. Extradural and intradural metastatic lesions may also mimic SEA (12–15).

Venous engorgement with enhancement can be observed both above and below an SEA. The enhancement may be due to direct extension of an inflammatory process along the venous plexus, mechanical obstruction of venous drainage (16,17), or both. We recently reviewed MR images of 450 subjects, including 35 healthy volunteers, in whom postcontrast MR imaging was performed to analyze visualization and dilatation of the spinal epidural veins (18). There was a statistically significant prevalence of venous dilatation among the cohort of patients who had epidural lesions (eg, SEA and metastases) and spinal canal stenosis due to degenerative disease and disk herniation. However, dilatation of the anterior epidural veins was noted in the upper cervical region in approximately 20% of the patients who did not have any abnormality in the spinal canal and in the volunteers. Therefore, care should be taken in image interpretation since such dilatation of the anterior epidural veins mimics an SEA when it is near areas of diskitis or osteomyelitis (18–20). In these instances, axial views help distinguish normal dilatation of the anterior epidural veins, which appears as symmetric enhancement with sparing of the posterior longitudinal ligament (Fig 5).

SEA often causes significant spinal cord compression that necessitates decompression. A complete neurologic deficit may be fully reversible with early aggressive treatment. Nonoperative medical treatment is also justified for poor surgical candidates. In the absence of osteomyelitis, at least 4 weeks of intravenous antibiotic therapy appears to be necessary. When osteomyelitis is present, at least 8 weeks of antibiotic treatment usually is required (1,2).

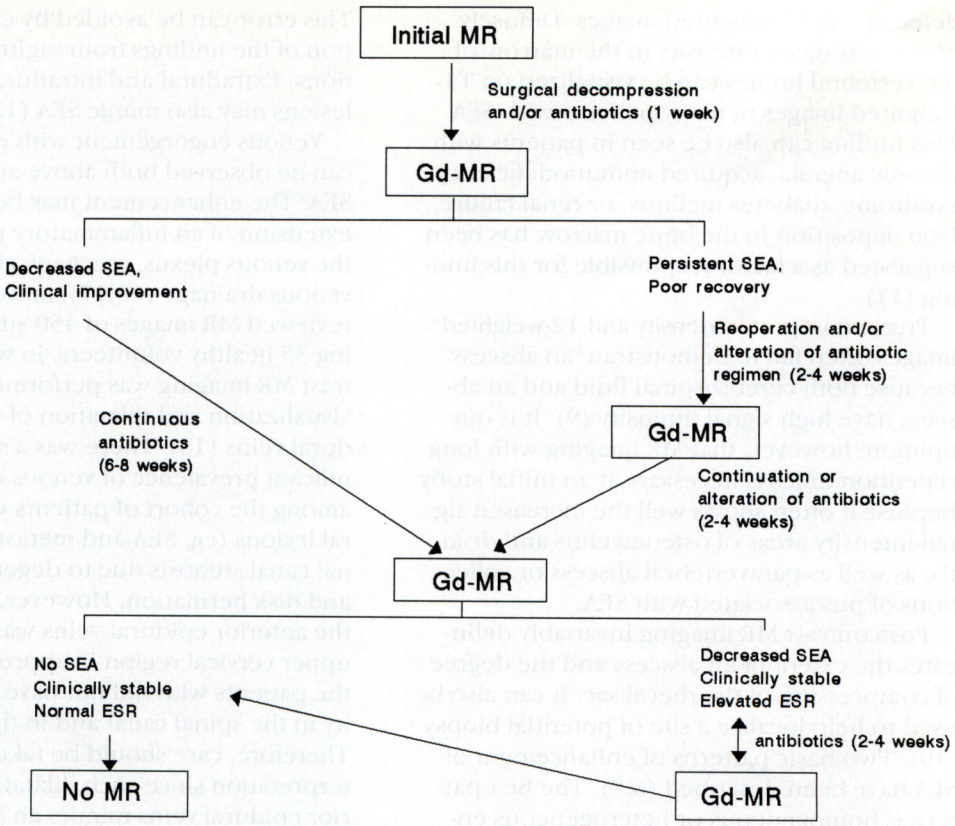


Figure 9. Algorithm of our institution's approach to MR imaging of SEA. *ESR* = erythrocyte sedimentation rate, *Gd-MR* = gadolinium-enhanced MR imaging.

Follow-up postcontrast MR imaging is important during the treatment of SEA because it can clearly demonstrate changes in the size of the abscess and the degree of thecal sac compression. The MR imaging approach during treatment of SEA at our institution is shown in Figure 9.

Since a decrease in the size of SEA was observed as early as 5 days, performance of follow-up MR imaging in 1 week to 10 days would be appropriate for evaluation of the initial response to antibiotic treatment or decompressive surgery. Patients with poor recovery from neurologic deficits are candidates for an immediate follow-up examination, followed by a second operation or alteration of antibiotic regimen and further follow-up examinations. Follow-up MR imaging examinations should be limited to pre- and postcontrast T1-weighted sagittal images to reduce the time and cost of the studies. Axial postcontrast images can be obtained if the findings on sagittal images are equivocal and recurrence of infection is suspected.

Enhancement of concomitant infectious foci adjacent to abscesses or sites of surgery can persist despite the resolution of an SEA and the absence of clinical evidence of infection. This persistent contrast enhancement most likely represents chronic sterile granulomatous changes or fibrosis (9,21) and does not necessitate continued antibiotic therapy. Close medical observation is recommended, however, especially if the patient has predisposing factors, since SEA may recur. Because the erythrocyte sedimentation rate was elevated uniformly among our patients during the active phase of SEA, serial measurements of erythrocyte sedimentation rate in addition to MR imaging will provide a helpful guide for determining appropriate duration of antibiotic therapy.

■ CONCLUSION

Gadolinium-enhanced MR imaging has proved to be indispensable for diagnosis of SEA. Close correlation of findings from sagittal and axial images is important to define the actual location and extent of disease. Follow-up imaging in both the early and late phases demonstrates good correlation with the clinical picture and is mandatory for optimal treatment planning in patients with SEA.

■ REFERENCES

1. Hlavin ML, Kaminski HJ, Ross JS, Ganz E. Spinal epidural abscess: a ten year perspective. Neurosurgery 1990; 27:177–184.
2. Danner RL, Hartman BJ. Update of spinal epidural abscess: 35 cases and review of the literature. Rev Infect Dis 1987; 9:265–274.
3. Angtuaco EJC, McConnell JR, Chadduck WM, Flanigan S. MR imaging of spinal epidural sepsis. AJNR 1987; 8:879–883.
4. Post MJD, Quencer RM, Montalvo BM, Katz BH, Eismont FJ, Green BA. Spinal infection: evaluation with MR imaging and intraoperative US. Radiology 1988; 169:765–771.
5. Bertino RE, Porter BA, Stimac GK, Tepper SJ. Imaging spinal osteomyelitis and epidural abscess with short T1 inversion recovery (STIR). AJNR 1988; 9:563–564.
6. Post MJD, Sze G, Quencer RM, Eismont FJ, Green BA, Gahbauer H. Gadolinium-enhanced MR in spinal infection. J Comput Assist Tomogr 1990; 14:721–729.
7. Sharif HS, Clark DE, Aabed MY, et al. Granulomatous spinal infections: MR imaging. Radiology 1990; 177:101–107.
8. Gero B, Sze G, Sharif H. MR imaging of intradural inflammatory diseases of the spine. AJNR 1991; 12:1009–1019.
9. Sandhu FS, Dillon WP. Spinal epidural abscess: evaluation with contrast-enhanced MR imaging. AJNR 1992; 12:1087–1093.
10. Kricun R, Shoemaker EI, Chovanes GI, Stephens HW. Epidural abscess of the cervical spine: MR findings in five cases. AJR 1992; 158:1145–1149.
11. Geremia GK, McCluney KW, Adler SS, et al. The magnetic resonance hypointense spine of AIDS. J Comput Assist Tomogr 1990; 14:785–789.
12. Sze G, Krol G, Zimmerman RD, Deck MDF. Malignant extradural spinal tumors: MR imaging with Gd-DTPA. Radiology 1988; 167:217–223.
13. Dillon WP, Norman D, Newton TH, Bolla K, Mark A. Intradural spinal cord lesions: Gd-DTPA–enhanced MR imaging. Radiology 1989; 170:229–237.
14. Sze G, Abramson A, Krol G, et al. Gadolinium-DTPA in the evaluation of intradural extramedullary spinal disease. AJNR 1988; 9:153–163.
15. Lim V, Sobel DF, Zyroff J. Spinal cord pial metastases: MR imaging with gadopentetate dimeglumine. AJNR 1990; 11:975–982.
16. Kaiser MC, Capesius P, Roilgen A, Sandt G, Poos D, Gratia G. Epidural venous stasis in spinal stenosis: CT appearance. Neuroradiology 1984; 26:435–438.
17. Ross JS, Modic MT, Massaryk TJ, Carter J, Marcus RE, Bohlman H. Assessment of extradural degenerative disease with Gd-DTPA-enhanced MR imaging: correlation with surgical and pathologic findings. AJNR 1990; 10: 1243–1249.
18. Sato S, Mihara F, Numaguchi Y, Rothman MI, Kawaguchi H. Spinal epidural venous plexus: MR enhancement pattern and clinical significance. Presented at the 30th Annual Meeting of the American Society of Neuroradiology, St Louis, Mo, May 31–June 5, 1992.
19. Breger RK, Williams AL, Daniels DL, et al. Contrast enhancement in spinal MR imaging. AJNR 1989; 10:633–637.
20. Gelber ND, Ragland RL, Knorr JR. Gd-DTPA enhanced MRI of cervical anterior epidural venous plexus. J Comput Assist Tomogr 1992; 16:760–763.
21. Ross JS, Delamarter R, Hueftle MG, et al. Gadolinium-DTPA-enhanced MR imaging of the postoperative lumbar spine: time course and mechanism of enhancement. AJNR 1989; 10:37–46.

Invited Commentary

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Several important points on the imaging and clinical management of SEA are raised by the preceding article by Numaguchi et al. Certainly, it is well accepted that MR imaging has replaced myelography and CT in the evaluation of potential infection of the spine, and it comes as no surprise that gadolinium-enhanced MR imaging is the best means of detecting the extent of SEA. Variable enhancing patterns dependent on the age of the infection are well known. However, a number of key points need to be stressed: (*a*) that T2-weighted images may be insensitive to the presence of SEA because both cerebrospinal fluid and the abscess may be hyperintense; (*b*) that the abscess may be spread widely

within the spinal canal in both craniocaudal and anteroposterior directions, even in the absence of localizing clinical signs; and (*c*) that subtle changes in the signal intensity of cerebrospinal fluid on T1-weighted images may reflect widespread involvement, compression of the thecal sac, and possible associated meningitis.

Underlying causes of SEA include those entities mentioned in the article. In addition, SEA may develop as a complication of surgery (eg, laminectomy or diskectomy). Appreciation of the fact that enhancement of granulation tissue can be seen normally after surgery helps prevent misdiagnosis. The appearance of enhancement of this scar tissue depends, however, on the interval from surgery to imaging. Specifically, if the patient is imaged early in the postoperative period, one might expect to see less enhancement of immature granulation tissue than of an inflammatory process. Later in the postoperative period, enhancement of more mature granulation tissue is differentiated from that of infection because enhancing scar tissue tends to be more localized to the operative site, whereas an infectious process such as SEA will spread away from the operative site.

Whatever the underlying cause of SEA, the decision to operate on an emergent basis or to use only antibiotic therapy after identification of the offending organism is clearly based on the patient's clinical status, specifically whether there are progressive neurologic symptoms. Surgery on certain patients, even in the presence of progressive symptoms, can be quite formidable, particularly those with an abscess in the anterior epidural space throughout many segments of the spine (as in Fig 2). Focal SEAs, particularly when posterior in location (as in Fig 3), are easier to drain and are associated with less postoperative morbidity. From an operative standpoint, exposure through laminectomy should extend to the most superior and most inferior part of the SEA. Adequate external drainage of the infection via an indwelling catheter and monitoring the success of this drainage with intraoperative sonography are crucial in surgical management. Sonography clearly shows whether loculated areas of abscess have collapsed. In addition, the nidus of the infection must be attacked if it can be identified (eg, diskitis, retroperitoneal abscess). Postoperatively, monitoring the erythrocyte sedimentation rate most closely tracks the efficacy of medical therapy (6 weeks or more of intravenous antibiotics).

When infection is clinically considered a possibility, MR imaging should be performed before and after gadolinium injection, both during the initial work-up and at follow-up. Preoperative images show the full extent of the abscess and may indicate the site of origin of the infection (eg, diskitis, paravertebral abscess). Radionuclide studies with gallium can add significantly to the patient's initial work-up, particularly when the findings (both clinically and radiologically) are subtle for infection. These studies not only tend to strengthen the diagnosis of infection but indicate the extent of the involvement, which may be underestimated on MR images.

MR imaging of multiple segments of the spine is clearly important when SEA is suspected. This means either a long study, with the placement of surface coils at the different spinal levels (cervical, thoracic, lumbar), or preferably the use of a multicoil (phase-array coil), which can image at least two segments of the spine at a time. An MR technique worth pursuing but not demonstrated in this article is fat-suppressed gadolinium-enhanced MR imaging. This technique allows the differentiation of fat from gadolinium-enhancing abnormalities such as SEA and is a recommended additional sequence when the available MR imager produces high-quality fat-suppressed images.

Follow-up imaging of SEA is crucial, whether the treatment has been surgical or nonsurgical. It is well appreciated that continued abnormal enhancement does not necessarily indicate an ongoing or suboptimally treated infection. Healed areas of prior infection may enhance due to the presence of scar tissue. The resolution of previously noted anatomic distortion is a helpful MR imaging finding that indicates a favorable medical response to treatment. Radionuclide studies are also used in assessing the efficacy of medical treatment.

SEA often manifests with total or severe paralysis but is one of the few abnormalities for which proper treatment can result in complete neurologic recovery. The variability in MR imaging appearance of SEA must be recognized, since it will help ensure appropriate treatment of this potentially devastating infection. Recognition of the different MR patterns of SEA (as demonstrated in this article) can hasten proper treatment.