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Epidural infection: Is it really an abscess?

Frank R. Avilucea, Alpesh A. Patel[1]

Department of Orthopaedic Surgery, University of Utah, Salt Lake City, Utah, [1]Department of Orthopaedic Surgery, Loyola University, Chicago, Illinois, USA

E-mail: Frank R. Avilucea - frank.avilucea@hsc.utah.edu; *Alpesh A. Patel - alpesh2@gmail.com
*Corresponding author

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Abstract

Background: We reviewed the literature regarding the pathogenesis, clinical presentation, diagnosis, and management of spinal epidural abscess (SEA).

Methods: Utilizing PubMed, we performed a comprehensive review of the literature on SEAs.

Results: SEA remains a difficult infectious process to diagnose. This is particularly true in the early stages, when patients remain neurologically intact, and before the classic triad of fever, back pain, and neurologic deficit develop. However, knowledge of risk factors, obtaining serologic markers, and employing magnetic resonance scans facilitate obtaining a prompt and accurate diagnosis. In patients without neurologic deficits, lone medical therapy may prove effective.

Conclusions: More prevalent over the previous three decades, SEA remains a rare but deleterious infectious process requiring prompt identification and treatment. Historically, identification of SEA is often elusive, diagnosis is delayed, and clinicians contend that surgical debridement is the cornerstone of treatment. Early surgery leads to more favorable outcomes and preserves neurologic function, particularly in the early stages of disease when minimal or no neurologic deficits are present. The advent of improved imaging modalities, diagnostic techniques, and multidrug antimicrobial agents has enabled medical/spinal surgical consultants to more rapidly diagnose SEA and institute more effective early medical treatment (e.g., data suggest that lone medical therapy may prove effective in the early management of SEA).

Key Words: Spine, epidural, abscess, infection

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INTRODUCTION

Spinal epidural abscess (SEA), as a bacterial infection of the spine resulting in accumulation of purulent fluid in the epidural space, has the potential to expand and compress the spinal cord. Depending upon the spinal level involved, the major feared catastrophic complications may include quadriplegia or paraplegia. Therefore, within the medical literature, SEA has classically been regarded as a surgical emergency, requiring decompression to preserve or improve neurologic status, and maintain spinal stability.

Nevertheless, the surgical recommendations are typically based upon expert opinion, retrospective studies, and case series. Despite the dogma to surgically intervene on these infections, there is a growing body of evidence demonstrating that a patient with minimal or no neurologic findings may respond well to appropriate

antimicrobial therapy alone.[3,46] The dilemma, however, is that although imaging modalities have drastically improved the ability to accurately diagnose SEA, the variable clinical presentation of SEA may delay establishing the correct diagnosis, resulting in suboptimal treatment, and contributing to the need for surgical debridement. In this review, we discuss the epidemiology, pathogenesis, clinical features, treatment, and outcomes of bacterial SEA, and whether additional clinical findings may hasten diagnosing and treating SEA.

EPIDEMIOLOGY

In 1975, Baker *et al*. reported an SEA incidence of 0.2–1.2 per 10,000 hospital admissions per year.[2] Since then, numerous reports document an increasing incidence: reports between 1992 and 2006 state an incidence of 2.5–3 per 10,000 hospital admissions.[7,12-14,23,35,42,43,47] The increased recognition of this infection likely relates to the improved accuracy in diagnoses imparted by magnetic resonance imaging (MR).[1,6,18] This trend in the United States is also reflective of an aging population with predisposing conditions or risk factors, such as diabetes mellitus, immunosuppressive therapy, cancer, human immunodeficiency virus (HIV), intravenous drug use, and renal failure.[2,12,14,23,35,41,47] Additionally, the increased use of epidural procedures for anesthesia or pain control likely contributes to these overall numbers despite the low 0.001% incidence of epidural abscess following catheter insertion.[51] Lastly, there are reports documenting occurrence at any age, however, the greatest prevalence occurs in the fifth to seventh decade of life, with a male-to-female ratio of 1:1.[12]

HISTORIC PERSPECTIVE: EPIDURAL SPINE INFECTIONS

Albers is credited with the first report of SEA, and in 1853, Duckeck termed this condition "peripachymeningitis," which by later reports was identified as "pachymeningitis externa."[11] In 1926, Dandy provides the first thorough review of the condition, its pathogenesis, as well as his case series where he documents a mortality rate of 81%.[11] In 1948, Heusner delineated the classical clinical features of SEA, which remain valid today.[22]

Sine qua non approach to surgical epidural abscess management

In the early part of the 20th century, the *sine qua non* approach to SEA management was immediate laminectomy for spinal decompression. The 20th century brought in the antibiotic era. Sulfonamide was developed by Gerhard Domagk in 1935, penicillin was discovered by Alexander Fleming in 1929, and clinically applied by Florey and Chain in 1940. Since then, a tremendous number of antibiotics have been developed to control wound infection. Heusner further reported a survival rate of 63% with surgery alone compared with 90% in the series of patients who received concomitant antibiotics. Aided by improvements in diagnosis and antibiotic treatment, Baker later reports a mortality of 18% in a series of 39 patients and Reihsaus identifies a mortality rate of 16% in meta-analysis of 915 patients.[2,41]

At present, numerous proponents recommend urgent surgical decompression as the treatment of choice for SEA.[10,12,14,22,23,31,37,42,47] As later discussed in this review, there is recent evidence, however, suggesting surgical decompression and debridement is necessary only when there is sepsis or burgeoning neurologic deficit.

EPIDURAL INFECTION PATHOGENESIS

Anatomic features

The vascular as well as the morphologic anatomy of the spinal canal and dura mater play a role in determining the evolution and anatomic features of SEA.

In fetal life, vascular channels traverse the endplates and begin to diminish in size at birth until complete disappearance by 5 years of age. In adults, the blood supply to the disc arises from two capillary plexuses: one penetrates 1–2 mm into the outer annulus, supplying only the periphery of the annulus. The second begins in the vertebral body and penetrates the subchondral bone terminating in capillary loops at the bone–cartilage interface. The capillary network density at this junction is greatest in the center and least at the periphery. In their 1959 report, Wiley *et al*. eloquently demonstrate that spinal arteries enter the canal through the intervertebral foramen.[52] Arterial branches ascend and descend to supply vertebral bodies cranially and caudally culminating in rich arterial anastamosis residing within the vertebral body metaphyseal region. Injection studies demonstrated that bacteria could easily spread hematogenously to these metaphyseal regions. These anatomic findings led to the arteriolar theory for hematogenous dissemination whereby bacteria may become lodged in the low-flow end-arteriolar arcade leading to establishment of infection that may result in not only vertebral osteomyelitis and discitis, but also SEA formation.

Venous theory for bacterial dissemintation

There is also a venous theory for bacterial dissemination. Through dye injection studies, Batson demonstrated that flow from the pelvic venous plexus to the vertebral venous plexus occurs via a valveless system and transpires with increased lower abdominal pressure or Valsalva, and is transmitted to the spinal thecal sac.[4,5] As the distribution of veins within the vertebral body is an aborization of vessels, Batson's findings provide another significant hematogenous mechanism in the establishment of an infectious focus in the spinal column.

Morphology of epidural space

The epidural space is lined with mesenchymal epithelium and is filled with loose adipose and areolar connective tissue, lymphatics, small arteries, and the epidural venous plexus. This space surrounds the dural sac and is bounded by the posterior longitudinal ligament ventrally, the ligamenta flava and lamina dorsally, and the pedicles of the spinal column and the intervertebral foramina and their neural elements laterally. Cranially, the space is anatomically closed at the foramen magnum where dura attaches with the endosteal dura of the cranium. Caudally, the epidural space terminates at the sacral hiatus, which is closed by the sacrococcygeal ligament.

The dimensions of this space are largely determined by variations in the spinal canal size. Ventrally, the dura abuts the canal from C1 to S2. Dorsally, however, the space begins to appear at C7 and gradually expands along the thoracic region to a depth between 0.5 and 0.75 cm between T4 and T8. The space tapers between T11 and L2 and thereafter attains its greatest depths below L2. Caudal to S2, the epidural space is present circumferentially.[8,9,24] It is plausible that the location and extent of SEA is associated with the anatomic confines of the spinal canal.

SEA is often localized in the posterior space as a true SEA and, if found anteriorly, is often associated with vertebral osteomyelitis.[12,23,47] Because the epidural space is a vertical sheath, abscesses that begin at one level commonly extend to multiple levels; studies show an average of three spinal segments.[25] Finally, as found in several studies, SEA is predominantly identified in the thoracic and lumbosacral region.[17,32,41,49,25]

Etiology/pathogenesis

Bacteria gain access to the epidural space via hematogenous dissemination from a distant site, contiguous spread from an infected neighboring structure, such as a retropharyngeal or psoas abscess, or iatrogenic inoculation. In 30–40% of cases, the source of infection is not identified.[12-14,34] Skin, soft-tissue, urinary, and respiratory tract infection are often the primary sources of infection. As the vertebral column is highly vascularized throughout its length, hematogenous dissemination may result in discontinuous SEA sites, which should be considered when assessing for spinal tenderness and planning imaging studies.

As most conditions allow for invasion of skin flora, *Staphylococcus aureus* is identified in approximately two-thirds of cases.[41,42,25] Microbes identified in the setting of spinal procedures or catheter placement include coagulase-negative staphylococci, such as *S. epidermidis*, as well as methicillin resistant *S. aureus*, which carries a particularly high risk and manifests within a few weeks after surgical intervention or spinal injection. Less commonly identified pathogens included gram-negative bacteria, especially *Escherichia coli*, often secondary to urinary tract infections, and *Pseudomonas aeruginosa*, which is often found in injection drugs users.[23,27,37,43] There remains debate among authors regarding the cause for neurologic impairment: some contend the impairment is due to vascular insult, whereas others support a mechanical compression etiology.[19]

CLINICAL PRESENTATION

Four-staged system to identify SEA

In an attempt to describe the clinical characteristics and severity of SEA, Heusner described a four-staged system [Table 1] to identify SEA: stage I, back pain, fever, and tenderness to palpation; stage II, radicular pain, nuchal rigidity/neck stiffness; stage III, neurologic deficits and bowel and bladder dysfunction; and stage IV, paralysis.[22,38] Without any form of treatment, patients will progress through these four stages. From a diagnostic standpoint, however, the progression from one stage to the next is highly variable and unpredictable. A patient may transition to weakness or paralysis in a few hours, or not develop any neurologic deficits for several months.[12,39]

Most common presenting symptoms

The most common presenting symptoms include back pain (85%), fever (50%), and neurologic deficit (32%).[41,42] This classic triad of symptoms, however, presents in the minority of patients.[10,15,22] The highly variable presentation, which causes initial misdiagnosis in half of cases leads to delay in treatment from the time of presentation and definitive treatment.[12] In a recent report by Huang *et al*., 79% of patients had received medical care for greater than 14 days after symptom onset and prior to arriving at the appropriate treatment.[25]

Diagnostic delay of SEA resulting in delayed treatment

The difficulty of diagnosing SEA often results in delay of diagnosis, which portends worse patient outcome. In their retrospective study, Davis *et al*. report upon the impact of delayed diagnosis on patient outcome in 47 patients: neurologic deterioration occurred in 57%, and 45% discharged with residual weakness compared with 13% without such delay.[15] The difficulty of diagnosing SEA leading to significant delay in treatment emphasizes

Table 1: Clinical Diagnosis of spinal epidural abscess

Stage	Clinical Findings
I	Back pain, fever, tenderness to palpation
II	Spinal root findings: radicular pain, nuchal rigidity, hyper-reflexia
III	Sensory findings, motor weakness, bowel/bladder dysfunction
IV	Paralysis

Stages of neurologic progression as initially described by Heusner.[22]

the importance of frequent neurologic examinations. Moreover, as the classic triad of symptoms is present in only 9% of patients, a more sensitive screen (98%) is to identify risk factors [Table 2] for bacteremia or direct inoculation of the epidural space.[15]

Systemic illness with acute spinal epidural abscess versus vertebral osteomyelitis

Patients with an acute SEA commonly have more systemic illness than those with vertebral osteomyelitis. In general, leukocytosis is identified in nearly two-thirds of patients and inflammatory markers, erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP), are consistently elevated [Table 3].[41,47] None of these laboratory abnormalities, however, are specific to SEA. In patients where blood has been assessed, bacteremia has been identified in 60% of patients, either as the cause or result of SEA.[10,12] Additionally, in patients where cerebrospinal fluid (CSF) is analyzed, high protein and a pleocytosis are found, findings suggestive of parameningeal inflammation but, again, not specific to SEA.[14] Finally, as found by Douriche *et al*., the results of gram staining CSF are often negative, and cultures are positive in less the one-quarter of patients.[14]

IMAGING

Radiographs show late changes for sea

Radiography is readily available, relatively inexpensive in screening and follow-up of patients, and often helpful in interpreting additional diagnostic imaging studies. Unfortunately, radiography fails to provide early evidence for infection in the setting of osteomyelitis and, as a projectional image, yields no clear evidence of an isolated epidural abscess. As radiography is unable to delineate the early stages of infection, advanced imaging studies are utilized to diagnose spinal infection.

Enhanced MR study of choice to identify spinal epidural abscess

MR is the proven study of choice to identify spinal infection and is reported to be 95% accurate.[1,6,18] As a noninvasive and safe imaging modality, MR with and without gadolinium delineates the extent and severity of spinal cord compression, as well as the extent of abscess in all directions. Moreover, it has the capability to identify disc space infection as well as osteomyelitis.

Table 2: Risk Factors for spinal epidural abscess

Risks Factor Associated with increased spinal epidural abscess prevalence
Intravenous drug use
Immunocompromized
Alcohol abuse
Recent spine procedure
Distant site infection
Diabetes
Indwelling catheter

Previous work shows that the finding of two or more risk factors is associated with a 98% prevalence of SEA.[15] While none of these factors in tandem are specific to SEA, epidural infection should remain highly considered in patients with an unknown source for suspected infection

Enhanced MR with osteomyelitis

In the setting of vertebral osteomyelitis, which may be directly involved in the evolution of a SEA, vertebral T2-weighted signal intensity increases due to associated edema, whereas T1-weighted signal intensity will decrease due to replacement of marrow fat by edematous fluid. A third commonly used pulse sequence, short tau inversion recovery (STIR), suppresses the bright signal from adipose tissue enabling lesions with relatively high water content (e.g., edema) to have increased signal.[16,26,50] The STIR sequence is highly sensitive for abnormalities, with a negative predictive value approaching 100% for acute osteomyelitis.[16,26,50]

MR characteristics for epidural fluid collections

The MR characteristics of an epidural abscess are high signal on T2-weighted images and low signal on T1-weighted images. Two types of enhancement have been described.[20] A homogenous enhancement is seen in abscesses with inflammatory tissue without purulent fluid. A true abscess, however, has been described to show only peripheral enhancement as the necrotic center of the abscess is not perfused and is a relatively inaccessible extravascular space with low accumulation of contrast material.[44] Precontrast T2-weighted images typically fail to show a SEA as both CSF and abscess formation show minimal contrast difference.[29,40,42] Precontrast T1-weighted images, however, may be helpful to identify subtle changes seen in CSF, which may suggest thecal sac compression, associated meningitis, and degree of involvement. A summary of MR findings is seen in Table 4.

Additional patterns of MR enhancement for spinal epidural abscess

Two additional patterns of enhancement may be present: linear enhancement along the dura and engorgement of the epidural or basivertebral veins. Linear enhancement represents extension of inflammation into the dura, and venous engorgement is observed above and below a SEA, which is the result of inflammatory extension along the venous plexus resulting in mechanical obstruction of venous drainage.[36] Sagittal MR of the entire spine is

Table 3: Laboratory Diagnosis of spinal epidural abscess[41, 47]

	Marker	Level
Serology	Blood cultures	Positive 60% of cases
	Leukocyte count	Elevated
	C-Reactive protein	Elevated
	Erythrocyte sedimentation rate	Elevated

recommended to identify the span of an abscess as well as localizing potential skip lesions.

Necessity for follow-up enhanced MR imaging

Previous studies report upon the necessity of follow-up imaging to assess efficacy of surgical or medical treatment. It has been reported that increased or diminished intensity of contrast enhancement at the site of a SEA correlates well with clinical deterioration or improvement, respectively.[36] In another study, Gillams *et al*. report that a high signal intensity rim at the lesion seen on T1-weighted images is one of the earliest findings suggestive of healing. They also report that gadolinium may increase the degree and extent of enhancement in some patients, a finding which does not indicate deterioration or treatment failure.[21] A recent study by Kowalski *et al*., however, finds that the use of serial MRs to assess for interval change after initiating antibiotic treatment does not correlate with clinical improvement and, therefore, should not be utilized to predict treatment failure.[28] The absence of known MR findings consistent with positive response to treatment, either surgical or medical, should dissuade physicians from obtaining serial MRs to assess for treatment efficacy, particularly in the patient with no neurologic deficits and improving serologic inflammatory markers.

Utility of CT-myelography for patients unable to undergo MR examinations

In the patient where MR may not be completed (e.g., cardiac pacemaker), myelography may be employed. In the event that pus is encountered during needle insertion, a specimen is sent for culture with extreme care taken to avoid entering the thecal sac. Myelography should thereafter be completed at another level. At the time of myelography, CSF needs to be assessed for total cell count, glucose, protein, evidence of pleocytosis, as well as culture and sensitivity. The CSF findings generally reflect a parameningeal infection with markedly increased protein content and no bacteria unless there is an associated subdural abscess or meningitis.[2,27] If a computed tomography (CT) scan can be done expeditiously after the myelogram is performed, the degree of neural compression will be defined more accurately.

TREATMENT AND OUTCOMES

Goals of treatment for SEA

The goals of treatment are infection eradication, preservation of neurologic status, pain relief, prevention of neurologic deterioration, and maintaining vertebral column stability. At present, numerous authors contend surgical decompression is necessary as there are a small proportion of patients that will develop rapid neurologic decline despite initiation of appropriate antibiotic treatment. There are, however, select cases where nonoperative management is classically recommended: patients who are poor surgical candidates, the abscess spans a considerable length of the vertebral canal, or there is paralysis, which has persisted for more than three consecutive days.

Efficacy of medical management of SEA

To date, there is a growing body of evidence reporting upon the efficacy of lone medical management.[30,33,48] Recently, Savage *et al*. reported on the early clinical outcome of medically treated SEA and concluded that it is a viable alternative to surgery for select patients presenting with back pain alone or neurological symptoms that have been stable for over 72 hours.[45] Additionally, Siddiq *et al*. describe a series of 25 patients over a 14-year span treated with antibiotics alone resulting in comparable or greater rates of complete recovery or minimal residual motor weakness compared with patients who underwent surgical decompression.[46] The increased use of MR and heightened awareness of SEA enable early diagnosis in the course of disease optimizing the potential for medical management efficacy.

Identification of organisms responsible for SEA

For medical management to proceed effectively, identification of the organism is necessary and may be accomplished through blood cultures or a percutaneous biopsy along with drainage. Once cultures are obtained, intravenous antibiotics should be initiated promptly. To monitor initial patient response to treatment, serial neurologic examination is routinely completed and serologic monitoring is accomplished through daily complete blood count (CBC), erythrocyte sedimentation rate (ESR), and C-reactive protein (CRP) levels. Definitive antibiotic treatment is predicated upon culture and sensitivity results whereby daily parenteral treatment is administered for 3–4 weeks in the setting of lone SEA and for 6–8 week if there is also vertebral osteomyelitis.[2,12]

Treatment of SEA with neurological deficit

In the setting of SEA with associated neurologic deficit, the predominant posterior location of most SEA renders it amenable to surgical decompression through a laminectomy. The facet joints are left intact to maintain spinal

Table 4: Neuroradiological Diagnosis of spinal epidural abscess

MR Sequence	Enhance MR signal changes	Patterns of enhancement
T1-Weighted sequence	Decreased	
T2-Weighted sequence	Increased	Homogenous collection or rim-enhancement only
STIR	Increased in the setting of vertebral osteomyelitis	

Summary of key findings identified on enhanced-MR associated with SEA

Table 5: Options for spinal epidural abscess treatment

Neurologic status	Type of infection identified	Treatment	Monitoring response to treatment
No deficit	Abscess ± vertebral osteomyelitis	Parenteral antibiotics	1. Neurologic exams 2. CBC, ESR, CRP
Presence of neurologic deficit	Abscess only	Laminectomy, preserve facets	1. Neurologic exams 2. CBC, ESR, CRP
	Abscess and presence of vertebral osteomyelitis	Laminectomy and anterior Decompression; instrumentation may be needed for stabilization.	1. Neurologic exams 2. CBC, ESR, CRP

stability. In the presence of SEA secondary to vertebral osteomyelitis, decompression and debridement may be best completed with an anterior and posterior exposure enabling treatment of both osteomyelitis and the epidural infection; instrumentation and fusion may be necessary in these cases due to compromised spinal stability. Lastly, the use of a drain following primary closure, or delayed primary closure once serologic markers and temperature return to normal may be utilized as a modality to minimize fluid accumulation following decompression.[2,12] Treatment options are summarized in Table 5.

SUMMARY

The presence of neurologic deficit plays a predominant role in the treatment algorithm of SEA, particularly in early stages of the disease. The increased use of MR and heightened awareness of SEA enable early diagnosis in the course of disease and thus optimizes the potential for medical management efficacy. The rapidity by which antibiotics are initiated following either blood cultures or percutaneous biopsy has dramatically improved the prognosis for recovery and preservation of neurologic status such that open surgical decompression should be reserved for patients identified with neurologic deficits in the early stages of disease. In line with this notion, close clinical assessment monitoring for any neurologic deterioration is paramount in nonoperative management.

REFERENCES

1. Angtuaco EJ, McConnell JR, Chadduck WM, Flanigan S. MR imaging of spinal epidural sepsis. AJR Am J Roentgenol 1987;149:1249-53.
2. Baker AS, Ojemann RG, Swartz MN, Richardson EP Jr. Spinal epidural abscess. N Engl J Med 1975;293:463-8.
3. Bamberger DM. Outcome of medical treatment of bacterial abscesses without therapeutic drainage: Review of cases reported in the literature. Clin Infect Dis 1996;23:592-603.
4. Batson OV. The Function of the vertebral veins and their role in the spread of metastases. Ann Surg 1940;112:138-49.
5. Batson OV. The valsalva maneuver and the vertebral vein system. Angiology 1960;11:443-7.
6. Bertino RE, Porter BA, Stimac GK, Tepper SJ. Imaging spinal osteomyelitis and epidural abscess with short TI inversion recovery (STIR). AJNR Am J Neuroradiol 1988;9:563-4.
7. Bluman EM, Palumbo MA, Lucas PR. Spinal epidural abscess in adults. J Am Acad Orthop Surg 2004;12:155-63.
8. Bridenbaugh PO, Greene NM. Spinal (subarachnoid) neural blockade: Anatomy. In: Cousins MJ, Bridenbaugh PO, editors. Philadelphia: J.B. Lippincott Company; 1988.
9. Bromage P. Epidural analgesia. Philadelphia: Saunders; 1978.
10. Curry WT Jr, Hoh BL, Amin-Hanjani S, Eskandar EN. Spinal epidural abscess: Clinical presentation, management, and outcome. Surg Neurol 2005;63:364-71; discussion 371.
11. Dandy WE. Abscess and inflammatory tumors in the spinal epidural space (So-called pachymeningitis externa). Arch Surg 1926;13:477-94.
12. Danner RL, Hartman BJ. Update on spinal epidural abscess: 35 cases and review of the literature. Rev Infect Dis 1987;9:265-74.
13. Darouiche RO. Spinal epidural abscess. N Engl J Med 2006;355:2012-20.
14. Darouiche RO, Hamill RJ, Greenberg SB, Weathers SW, Musher DM. Bacterial spinal epidural abscess. Review of 43 cases and literature survey. Medicine (Baltimore) 1992;71:369-85.
15. Davis DP, Wold RM, Patel RJ, Tran AJ, Tokhi RN, Chan TC, *et al.* The clinical presentation and impact of diagnostic delays on emergency department patients with spinal epidural abscess. J Emerg Med 2004;26:285-91.
16. Erdman WA, Tamburro F, Jayson HT, Weatherall PT, Ferry KB, Peshock RM. Osteomyelitis: Characteristics and pitfalls of diagnosis with MR imaging. Radiology 1991;180:533-9.
17. Ericsson M, Algers G, Schliamser SE. Spinal epidural abscesses in adults: Review and report of iatrogenic cases. Scand J Infect Dis 1990;22:249-57.
18. Erntell M, Holtas S, Norlin K, Dahlquist E, Nilsson-Ehle I. Magnetic resonance imaging in the diagnosis of spinal epidural abscess. Scand J Infect Dis 1988;20:323-7.
19. Feldenzer JA, McKeever PE, Schaberg DR, Campbell JA, Hoff JT. The pathogenesis of spinal epidural abscess: Microangiographic studies in an experimental model. J Neurosurg 1988;69:110-4.
20. Friedmand DP, Hills JR. Cervical epidural spinal infection: MR imaging characteristics. AJR Am J Roentgenol 1994;163:699-704.
21. Gillams AR, Chaddha B, Carter AP. MR appearances of the temporal evolution and resolution of infectious spondylitis. AJR Am J Roentgenol 1996;166:903-7.
22. Heusner AP. Nontuberculous spinal epidural infections. N Engl J Med 1948;239:845-54.
23. Hlavin ML, Kaminski HJ, Ross JS, Ganz E. Spinal epidural abscess: A ten-year perspective. Neurosurgery 1990;27:177-84.
24. Hogan QH. Epidural anatomy examined by cryomicrotome section. Influence of age, vertebral level, and disease. Reg Anesth 1996;21:395-406.
25. Huang PY, Chen SF, Chang WN, Lu CH, Chuang YC, Tsai NW, *et al.* Spinal epidural abscess in adults caused by Staphylococcus aureus: Clinical characteristics and prognostic factors. Clin Neurol Neurosurg 2012;114:572-6.
26. Jones KM, Unger EC, Granstrom P, Seeger JF, Carmody RF, Yoshino M. Bone marrow imaging using STIR at 0.5 and 1.5 T. Magn Reson Imaging 1992;10:169-76.
27. Kaufman DM, Kaplan JG, Litman N. Infectious agents in spinal epidural abscesses. Neurology 1980;30:844-50.
28. Kowalski TJ, Berbari EF, Huddleston PM, Steckelberg JM, Osmon DR. Do follow-up imaging examinations provide useful prognostic information in patients with spine infection? Clin Infect Dis 2006;43:172-9.
29. Kuker W, Mull M, Mayfrank L, Topper R, Thron A. Epidural spinal infection.

Variability of clinical and magnetic resonance imaging findings. Spine (Phila Pa 1976) 1997;22:544-50; discussion 551.
30. Liem LK, Rigamonti D, Wolf AL, Robinson WL, Edwards CC, DiPatri A. Thoracic epidural abscess. J Spinal Disord 1994;7:449-54.
31. Lu CH, Chang WN, Lui CC, Lee PY, Chang HW. Adult spinal epidural abscess: Clinical features and prognostic factors. Clin Neurol Neurosurg 2002;104:306-10.
32. Mackenzie AR, Laing RB, Smith CC, Kaar GF, Smith FW. Spinal epidural abscess:The importance of early diagnosis and treatment. J Neurol Neurosurg Psychiatry 1998;65:209-12.
33. Mampalam TJ, Rosegay H,Andrews BT, Rosenblum ML, Pitts LH. Nonoperative treatment of spinal epidural infections. J Neurosurg 1989;71:208-10.
34. Maslen DR, Jones SR, Crislip MA, Bracis R, Dworkin RJ, Flemming JE. Spinal epidural abscess. Optimizing patient care.Arch Intern Med 1993;153:1713-21.
35. Mattle H, Jaspert A, Forsting M, Sieb JP, Hanny P, Ebeling U. [Acute spinal epidural abscess]. Dtsch Med Wochenschr 1986;111:1642-6.
36. Numaguchi Y, Rigamonti D, Rothman MI, Sato S, Mihara F, Sadato N. Spinal epidural abscess: Evaluation with gadolinium-enhanced MR imaging. Radiographics 1993;13:545-59; discussion 559-60.
37. Nussbaum ES, Rigamonti D, Standiford H, Numaguchi Y, Wolf AL, Robinson WL. Spinal epidural abscess:A report of 40 cases and review. Surg Neurol 1992;38:225-31.
38. Peterson JA, Paris P, Williams AC. Acute epidural abscess. Am J Emerg Med 1987;5:287-90.
39. Phillips GE, Jefferson A. Acute spinal epidural abscess. Observations from fourteen cases. Postgrad Med J 1979;55:712-5.
40. Post MJ, Sze G, Quencer RM, Eismont FJ, Green BA, Gahbauer H. Gadolinium-enhanced MR in spinal infection. J Comput Assist Tomogr 1990;14:721-9.
41. Reihsaus E, Waldbaur H, Seeling W. Spinal epidural abscess: A meta-analysis of 915 patients. Neurosurg Rev 2000;23:175-204; discussion 205.
42. Rigamonti D, Liem L, Sampath P, Knoller N, Namaguchi Y, Schreibman DL, *et al*. Spinal epidural abscess: Contemporary trends in etiology, evaluation, and management. Surg Neurol 1999;52:189-96; discussion 197.
43. Sampath P, Rigamonti D. Spinal epidural abscess: A review of epidemiology, diagnosis, and treatment. J Spinal Disord 1999;12:89-93.
44. Sandhu FS, Dillon WP. Spinal epidural abscess: Evaluation with contrast-enhanced MR imaging. AJNR Am J Neuroradiol 1991;12:1087-93.
45. Savage K, Holtom PD, Zalavras CG. Spinal epidural abscess: Early clinical outcome in patients treated medically. Clin Orthop Relat Res 2005;439:56-60.
46. Siddiq F, Chowfin A, Tight R, Sahmoun AE, Smego RA Jr. Medical vs surgical management of spinal epidural abscess. Arch Intern Med 2004;164:2409-12.
47. Soehle M, Wallenfang T. Spinal epidural abscesses: Clinical manifestations, prognostic factors, and outcomes. Neurosurgery 2002;51:79-85; discussion 86-7.
48. Sorensen P. Spinal epidural abscesses: Conservative treatment for selected subgroups of patients. Br J Neurosurg 2003;17:513-8.
49. Tang HJ, Lin HJ, Liu YC, Li CM. Spinal epidural abscess-experience with 46 patients and evaluation of prognostic factors. J Infect 2002;45:76-81.
50. Unger E, Moldofsky P, Gatenby R, Hartz W, Broder G. Diagnosis of osteomyelitis by MR imaging. AJR Am J Roentgenol 1988;150:605-10.
51. Wang LP, Hauerberg J, Schmidt JF. Incidence of spinal epidural abscess after epidural analgesia: A national 1-year survey. Anesthesiology 1999;91:1928-36.
52. Wiley AM, Trueta J. The vascular anatomy of the spine and its relationship to pyogenic vertebral osteomyelitis. J Bone Joint Surg Br 1959;41-B:796-809.

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