Spinal epidural abscess

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INTRODUCTION

Epidural abscess is a rare but important suppurative infection of the central nervous system. Abscesses that are enclosed within the bony confines of the skull or spinal column can expand to compress the brain or spinal cord and cause severe symptoms, permanent complications, or even death. Prompt diagnosis and proper treatment can avert complications and achieve cure in many cases. Both the diagnosis and management of epidural abscess, which often includes a surgical procedure for aspiration or drainage of the abscess, have been greatly aided by the advent of modern imaging techniques, such as computed tomography and especially magnetic resonance imaging.

The epidemiology, microbiology, clinical manifestations, diagnosis, and treatment of spinal epidural abscess (SEA) will be reviewed here. Intracranial epidural abscess, brain abscess, and bacterial meningitis are discussed separately. (See "Intracranial epidural abscess" and "Pathogenesis, clinical manifestations, and diagnosis of brain abscess" and "Treatment and prognosis of bacterial brain abscess" and "Epidemiology of community-acquired bacterial meningitis in adults" and "Pathogenesis and pathophysiology of bacterial meningitis" and "Clinical features and diagnosis of acute bacterial meningitis in adults" and "Initial therapy and prognosis of community-acquired bacterial meningitis in adults" and "Treatment of bacterial meningitis caused by specific pathogens in adults" and "Neurologic complications of bacterial meningitis in adults".)

TYPES OF EPIDURAL ABSCESS

Two distinct varieties of epidural abscess occur: SEA and intracranial epidural abscess (IEA). SEA is more common by a factor of nine to one. The distinction between these two entities is based upon the different anatomy of the two locations within the central nervous system and some differences in symptoms and natural history.

IEA is discussed in detail separately. (See "Intracranial epidural abscess".)

ANATOMY

The epidural space is the area between the dura mater and the vertebral wall. The anatomy of the spinal canal and dura mater determines many features of epidural abscesses [1]. For example, the dura is adherent to the bone above the foramen magnum. In contrast, an actual or true epidural space exists below the foramen magnum posterior and lateral to the spinal cord that extends down the length of the spinal canal (figure 1). This space is small in the cervical region and larger in the sacral region. The epidural space contains fat as well as arteries and a venous plexus. SEAs are most common in the thoracolumbar areas, where the epidural space is larger and contains more infection-prone fat tissue [2-4].

The majority of SEAs are located posteriorly; when anterior SEAs occur, they are usually below L1. Because the epidural space is a vertical sheath, abscesses that begin at one level commonly extend to multiple levels.

PATHOGENESIS

Bacteria can gain access to the epidural space hematogenously, by direct extension from infected contiguous tissue (such as a vertebral body or psoas muscle), or via direct inoculation into the spinal canal (eg, during spinal or epidural anesthetic procedures or surgery). Approximately one-third of patients with SEA have no identifiable source for the infection (table 1). Among the two-thirds for whom a portal of entry can be identified, the most common sites of origin are infections of skin and soft tissues and complications of spinal surgery or other invasive procedures, including epidural catheters that are left in place, usually for pain control.

SEA from epidural catheters can result from contamination at the time the catheter is inserted, by ascending contamination of the catheter from normal skin flora following catheter insertion,

from injection using contaminated syringes or local anesthetic solutions, or by hematogenous seeding from a bacteremia that occurs while the catheter is in place [5]. The importance of infection control measures to prevent epidural abscesses associated with epidural catheters was illustrated by a report in which the infecting organism was subsequently isolated from nasal swabs taken from the physician who placed the catheter [6]. This finding has prompted the recommendation to use face masks when epidural catheters are placed. Safe injection practices must also be used during spinal injection procedures in order to prevent bacterial contamination of medication vials [7].

Many SEAs begin as a focal pyogenic infection involving the vertebral disc or the junction between the disc and the vertebral body (pyogenic infectious discitis) (figure 1) [8,9]. As the pyogenic inflammation progresses and the abscess extends longitudinally in the epidural space, damage to the spinal cord can be caused by one or more of the following mechanisms [10]:

- Direct compression
- Thrombosis and thrombophlebitis of nearby veins
- Interruption of the arterial blood supply
- Bacterial toxins and mediators of inflammation

EPIDEMIOLOGY

Incidence — Overall, SEA is uncommon. As an example, in a retrospective study from 2004 to 2014 at a large academic hospital in the United States, the incidence of SEA was 5.1 cases per 10,000 admissions [11].

This incidence may represent an increase compared with prior decades [1]. During the period 1990 to 2000, the incidence in a county in Minnesota was 0.88 cases/100,000 person-years [12].

Part of this increased incidence may relate to the fact that the sensitivity and accuracy of diagnosis have been improved by the use of magnetic resonance imaging [13]. In the United States, the increasing incidence of SEA may also relate to the aging of the population and increases in the number of patients undergoing invasive spinal procedures for anesthesia or pain control.

The median age of onset of SEA is approximately 50 years, and the prevalence appears to be greatest between age 50 and 70 [14], but SEA can occur at any age. The incidence has been higher in males in some studies [14].

Predisposing factors and comorbid conditions — SEA can be a secondary complication of any condition that results in bacteremia [14]. As examples, injection drug use or dental abscesses, infected catheters, and infective endocarditis are all risk factors for SEA.

Spinal interventions can also result in SEA. Specifically, epidural catheter placement is an important risk factor. The reported incidence of SEA following placement of epidural catheters has ranged from 0.5 to 3 percent [15-18]. The risk of SEA is substantially lower when catheters are placed for short time periods, as occurs in obstetrical anesthesia [19]. Paraspinal injections of glucocorticoids or analgesics can also be complicated by the subsequent development of SEA [20].

SEAs are common in patients with vertebral osteomyelitis. In one report, 64 of 167 patients (38 percent) with hematogenous vertebral osteomyelitis due to methicillin-susceptible *Staphylococcus aureus* (MSSA) or gram-negative bacilli had radiographic evidence of SEA [21]. The incidence was significantly higher with MSSA than gram-negative bacilli (47 versus 29 percent).

Other possible risk factors for SEA include [22,23]:

- Alcoholism
- Diabetes
- HIV infection
- Trauma
- Tattooing
- Acupuncture
- Contiguous bony or soft tissue infection

MICROBIOLOGY

The leading bacterial pathogen causing SEA is *S. aureus*, which accounts for about two-thirds of cases caused by pyogenic bacteria [1-3,11,24,25]. Various other bacteria can also produce the infection. The approximate frequency of the leading causes of SEA (excluding mycobacteria) is as follows [2,10,24]:

- *S. aureus* – 63 percent
- Gram-negative bacilli – 16 percent
- Streptococci – 9 percent
- Coagulase-negative staphylococci – 3 percent, mostly occurring in patients with prior spinal instrumentation [3]

- Anaerobes – 2 percent
- Others (eg, fungi, parasites) – 1 percent
- Unknown – 6 percent

Mycobacterium tuberculosis is a more frequent cause of SEA in certain resource limited settings [26]. (See "Bone and joint tuberculosis".)

The proportion of *S. aureus* infections that are methicillin-resistant varies at different institutions and has ranged from 25 to 68 percent [11,22,27].

CLINICAL MANIFESTATIONS

The initial manifestations of SEA are often nonspecific and include fever and malaise. The classical diagnostic triad consists of fever, back pain, and neurologic deficits. However, only a small proportion of patients have all three components at presentation [27-29].

- Back pain is the most common symptom and is present in 70 percent to 100 percent of cases [30]. The duration of back pain prior to presentation ranges from 1 day to 2 months. Spinal tenderness has been found in 17 to 98 percent of cases [30].

- Fever is an important diagnostic clue, but is the classic finding most likely to be absent on presentation. As an example, only 23 of 48 patients (48 percent) were febrile in one case series reported from a single tertiary care center [29]. When fever is absent, it may lead to delayed or missed diagnosis [27].

- Neurologic deficits have been reported in up to half of the cases and include motor weakness, radiculopathy, and bowel and bladder dysfunction [30]. Even a small SEA can cause severe neurologic symptoms and sequelae. Untreated abscess can result in a typical progression of neurologic symptoms over time, starting with [3]:

  - Back pain, which is often focal and severe, then
  - Nerve root pain, described as "shooting" or "electric shocks" in the distribution of the affected nerve root, then
  - Motor weakness, sensory changes, and bladder or bowel dysfunction, and then
  - Paralysis

  Once paralysis develops, it may quickly become irreversible. Thus, urgent intervention may be required if progression of weakness or other neurologic findings are detected. (See

'Management' below.)

On direct examination, epidural abscesses may contain frank pus, especially in acute cases, but it is quite common to find only granulation tissue at the time of surgical drainage. Granulation tissue is often present when abscesses have been present for more than two weeks.

LABORATORY FINDINGS

Routine laboratory studies are seldom helpful in the diagnosis of SEA. The hematocrit is usually normal, and the leukocyte count may be elevated or normal. In a retrospective study of patients presenting to the emergency department, only 60 percent of patients who were diagnosed with SEA had leukocytosis at their initial visit [28].

The erythrocyte sedimentation rate (ESR) is elevated in nearly all cases of SEA and is often significantly elevated [31,32]. As with the ESR, the C-reactive protein (CRP) level is elevated in nearly all cases of SEA [30]. In the setting of back pain, an elevated CRP has been proposed as a clinical sign that should trigger a workup for SEA [33]. With this approach, use of CRP has been shown to shorten the time to diagnosis of patients with SEA. Although ESR and CRP can raise suspicion for SEA, they are nonspecific and are usually also elevated in other infections, including vertebral osteomyelitis in the absence of SEA.

DIAGNOSIS

Clinical suspicion — The diagnosis of SEA should be suspected in febrile patients with spinal pain accompanied by an examination consistent with a radiculopathy or other focal neurologic findings, especially if the pain is worsened by palpation or percussion. However, all three findings (eg, fever, pain and focal neurologic findings) are often not present initially. In patients who have only fever and back pain or only new-onset back pain, a low threshold for suspicion of SEA is warranted if risk factors are present (eg, injection drug use, chronic indwelling venous catheter, distant infected foci [eg, diabetic foot ulcer], older age, or recent spinal manipulation) [33].

Although routine laboratory studies are seldom helpful in the diagnosis of SEA, an elevated erythrocyte sedimentation rate (ESR) or C-reactive protein (CRP) in the setting of one or more typical symptoms of SEA should raise suspicion particularly if any risk factors are present.

Once the diagnosis of SEA is seriously considered, imaging of the spinal column is warranted and should be done as soon as possible. If the suspicion for SEA is high, the appropriate

medical team and surgical team (neurosurgery or orthopedic spine surgery) should be notified as soon as imaging findings reveal findings consistent with an SEA. (See 'Imaging to confirm diagnosis' below.)

Even though most patients with SEA have back pain, SEA is a rare cause of back pain. Thus, the diagnosis is infrequently made, or even considered, when patients with this condition first seek medical care. In fact, most patients present to health care providers multiple times before the diagnosis is made. A large retrospective database study that evaluated patients with a new diagnosis of SEA in the United States Department of Veterans Affairs in 2013 found that red flags (eg, unexplained weight loss, neurologic deficits, and fever) were often missed leading to delays in diagnosis [34]. Median time to diagnosis in these cases where epidural abscess was not initially considered despite the presence of red flags was 12 days, compared with 4 days in other cases.

Another study found that diagnostic delays were significantly less common during a five-year study period after a decision guideline was developed for all patients who sought care for spinal pain [31]. The decision guideline included a risk factor assessment for all patients initially lacking neurologic deficits, followed by ESR and CRP measurements prior to obtaining imaging studies. The risk factor assessment included obtaining information about the following factors: diabetes, history of injection drug use, chronic liver or kidney disease, recent spine procedures, spine surgery or fractures, indwelling vascular catheters, other site of infection, or an immunocompromising condition. Diagnostic delays were observed in 46 of 55 patients (84 percent) during a nine-year period prior to the institution of this decision guideline, compared with 3 of 31 patients (10 percent) during the five-year period following the use of the above decision guideline. It should be noted, however, that these data were part of a clinical study focusing on this problem and may not be reflective of day-to-day clinical practice even with the adoption of a decision guideline.

Imaging to confirm diagnosis — Spine imaging should be performed in all patients clinically suspected of having a SEA. In a patient with compatible clinical findings (eg, fever and back pain), identification of an epidural collection consistent with abscess is sufficient to make the presumptive diagnosis. Magnetic resonance imaging (MRI) with contrast (gadolinium) is the preferred imaging test because it is often positive early in the course of the infection and provides the best visualization of the location and extent of inflammatory changes (image 1) [25,35,36]. Careful examination of scout lateral images of the entire spinal column may be warranted even when patients have focal signs or symptoms in one region. Multiple skip lesions representing several levels of involvement may be seen, and patients may not have pain or tenderness in all of the affected areas. Rarely, repeat imaging in two to three weeks is indicated

in patients with an initially negative MRI if pain worsens or becomes more localized, if focal neurologic findings persist or worsen, and if an alternate diagnosis cannot be established [30].

Abscesses visualized by MRI usually demonstrate fluid-equivalent signal intensity on T2-weighted images with rim enhancement and a hypointense center [21,37]. Some SEAs are associated with ring-like enhancement or linear dural enhancement that some experts have suggested may be important indicators of the need for surgery [38,39]. Additional study is necessary to determine whether this is the case. If gadolinium cannot be used (eg, in patients with renal insufficiency), the presence of paraspinal and bone marrow edema are the most common findings in patients with SEA [40].

Longitudinal extension is common when infection arises in the epidural space. The average extent is three to five spinal cord segments [3], but the whole length of the spinal column may be involved.

Epidural abscesses can also occur uncommonly at noncontiguous locations in the spine. In a retrospective study at an academic tertiary care hospital, 22 of 233 adult patients (9 percent) with SEAs had at least two lesions in different anatomic regions of the spine [41]. Multifocal, noncontiguous SEAs were associated with delay in presentation (defined as the presence of symptoms for seven days or longer), a concomitant area of infection outside the spine or paraspinal region, and an erythrocyte sedimentation rate ≥95 mm/hour at presentation.

Although most SEAs arise spontaneously, they also may complicate the course of patients with primary vertebral osteomyelitis in which case the epidural abscess is more likely to occur in the lumbar region [42].

Distinguishing epidural soft tissue edema from epidural abscess is important. This distinction is primarily made by MRI. In contrast to the characteristic findings of abscess, soft tissue edema usually manifests as diffuse hypointensity of T1-weighted images, hyperintensity on T2-weighted images, and contrast enhancement. Epidural soft tissue edema also does not generally cause typical neurologic symptoms. Computed tomography (CT) scanning with intravenous contrast is an acceptable alternative to MRI if MRI is not immediately available or if it is contraindicated [3,43]. Plain radiographs of the spine may reveal changes of osteomyelitis or discitis but are rarely diagnostic of SEA. Myelography is now largely obsolete.

Identifying the pathogen — Once an intraspinal mass that could be an SEA has been identified, it is important to try to isolate the etiologic organism from samples of blood and abscess contents [1].

Two sets of blood cultures should be drawn in all patients with suspected SEA.

The approach to obtaining abscess specimens for microbiologic testing depends on the surgical plan, since not all patients undergo surgical debridement (see 'Deciding on surgical versus medical treatment' below):

- If a decision is made to proceed with surgical debridement, specimens for identifying the causative organisms can be collected at the time of surgery.
- If surgery is delayed or deferred, efforts should be made to obtain a sample for culture in order to target antibiotic therapy. The best specimen for culture is fluid or pus of the epidural abscess or inflammatory mass obtained by direct needle aspiration, usually using CT guidance.

Surgical specimens or aspirated fluid should be sent for aerobic and anaerobic bacterial culture and Gram stain, fungal and mycobacterial stains, and culture.

Lumbar puncture for cerebrospinal fluid (CSF) examination is typically not performed because the diagnostic yield is low and because of the risk of introducing infection into the central nervous system if the needle goes through an affected area. In most cases, the CSF findings are limited to a nonspecific elevation of protein and pleocytosis. CSF Gram stain is usually negative, and cultures are infrequently positive [3,22].

The approximate frequency of positive cultures from various sources in SEA is [10]:

- Abscess contents – 90 percent
- Blood – 62 percent
- CSF – 19 percent [3]

DIFFERENTIAL DIAGNOSIS

The differential diagnosis of SEA is extensive because many conditions cause back pain and neurologic deficits. These can be distinguished from SEA by radiological imaging. Important entities to consider in addition to SEA include:

- Disc and degenerative bone disease − These entities often present with back pain and may have focal neurological findings if the nerve root is involved. However, fever is not a typical presenting symptom.
- Metastatic tumors − Metastatic lesions may involve the spinal column with pain as the primary presenting symptom and in some cases, focal neurologic deficits depending on the location of the lesion. Fever is uncommon in this setting.

- Vertebral discitis and osteomyelitis − These entities often present with back pain and may have focal neurologic findings; fever may or may not be present. In certain instances, there may be an associated SEA. It is important to realize that *S. aureus* is a common cause of osteomyelitis and discitis as well as SEA; the combination of back pain and *S. aureus* bacteremia is not specific for SEA. [22]. (See "Vertebral osteomyelitis and discitis in adults", section on 'Symptoms and signs'.)

- Herpes zoster, prior to the appearance of skin lesions − Pain (acute neuritis) is the most common symptom of herpes zoster prior to the appearance of the rash, typically in a dermatomal distribution without focal neurologic findings or fever. (See "Epidemiology, clinical manifestations, and diagnosis of herpes zoster", section on 'Acute neuritis'.)

Common causes of low back pain in adults are discussed separately. (See "Evaluation of low back pain in adults".)

MANAGEMENT

The principles of optimal therapy of a SEA are reduction in size and ultimate elimination of the inflammatory mass and eradication of the causative organism. These goals are generally accomplished by a combination of surgical drainage and antibiotic therapy.

Antibiotics should be started as soon as the diagnosis of SEA is strongly suspected and immediately following the collection of two sets of blood cultures. (See 'Timing of initiation of antimicrobial therapy' below.)

Surgical decompression for most patients

Deciding on surgical versus medical treatment — Surgical decompression and drainage, in addition to systemic antibiotic therapy, is the treatment of choice for many patients. Surgical intervention is particularly important for those with acute or progressive neurologic deficits, spinal instability, ring-enhancing lesions on magnetic resonance imaging (MRI), or disease progression on antibiotic therapy.

Occasionally, a conservative medical only approach may be appropriate in highly selected patients who meet the following criteria:

- No risk factors for poor outcomes (eg, advanced age, diabetes mellitus, bacteremia, white blood cell counts >12,500 cell/L, and methicillin-resistant *S. aureus* [MRSA] infection).

- The infecting organism is known from aspirate cultures.

- There is no neurologic defect and no evidence of cord compression on MRI.

Medical therapy alone may also be appropriate if [14,39]:

- The patient refuses surgery.
- The patient is medically unstable and thus has an unacceptable surgical risk.
- The SEA has resulted in a complete spinal cord injury for greater than 48 hours and there is no evidence of an ascending spinal lesion.

Monitoring patients for whom surgery is deferred is discussed below. (See 'Approach if surgery is deferred' below.)

No randomized trials of medical (or conservative) versus surgical management of SEA exist.

There are a number of reports documenting recovery in patients with epidural abscesses following simple aspiration (for diagnosis) and antibiotic therapy alone without surgical decompression [44,45]. In most of these cases, the abscess was either not associated with any neurologic deficit or accompanied by minor weakness at the time of treatment.

One retrospective study involving 128 patients reported a failure rate of 41 percent in patients initially treated medically [44]. Diabetes, C-reactive protein (CRP) level >115 mg/L (11.5 mg/dL), white blood cell count >12,500 cells/L, and bacteremia were predictive of failure of medical therapy, which in this and most other studies was defined as a physician-documented decline in motor status while on intravenous antibiotics. Other studies have shown failure rates of medical therapy ranging from 31 to 75 percent [29,46-48]. These studies also suggested that other factors such as infection due to MRSA and age >65 years are additional risk factors for failure of medical therapy. It is important to note that several studies have shown medical therapy failure rates between 8.5 and 17 percent even in "ideal" patients who lacked the above risk factors [46,47].

Timing of surgery — Surgery should be performed as early as possible and within 24 to 36 hours of onset of paralysis [22].

In many cases, early surgical decompression and drainage are critical factors that will improve the ultimate prognosis [25,49]. In one retrospective study, 15 patients presented with neurologic deficit and underwent surgical decompression in addition to antibiotics [49]. A good outcome (eg, independent ambulation and continent of bowel and bladder) occurred in 80 percent of those who had surgical intervention within 24 hours compared to 10 percent in those who underwent decompression after 24 hours.

Approach if surgery is deferred — As above, we only choose a medical therapy approach in select patients (see 'Deciding on surgical versus medical treatment' above). When surgery is deferred, in addition to antibiotic therapy, a conservative approach includes the following steps:

- A careful neurologic monitoring strategy is identified prior to initiating treatment.
- A follow-up MRI should be performed to confirm diminishing size or resolution of the abscess. The timing of such follow-up imaging should be based on the clinical response of the patient by serial examinations and other routine indicators of a clinical response.
- Disclosure to patients that deterioration of neurologic function may occur while on medical therapy that will in turn require emergency surgical intervention.

Antimicrobial therapy

Timing of initiation of antimicrobial therapy — Antibiotics should be started as soon as the diagnosis of SEA is strongly suspected and immediately following the collection of two sets of blood cultures. We do not typically delay antibiotics in order to obtain a pretreatment specimen from the SEA (eg, abscess fluid) to confirm the diagnosis. In most cases, an imaging study (ideally MRI) can be obtained quickly and is sufficient to make a clinical diagnosis of SEA. If an imaging study is not immediately available and the suspicion for SEA is high, it is reasonable to start antibiotics while awaiting the imaging study. (See 'Management' above.)

Empiric therapy — Initial treatment regimens are usually empiric and designed to target the most common pathogens. An empiric regimen with antibiotics active against staphylococci (including MRSA), streptococci, and gram-negative bacilli should be chosen.

Appropriate empiric parenteral regimens include:

- Vancomycin (15 to 20 mg/kg intravenously [IV] every 8 to 12 hours for patients with normal renal function; maximum 2 g per dose initially, with a target serum trough concentration of 15 to 20 mcg/mL)

  PLUS

- Ceftriaxone (2 g IV every 12 hours)

An alternative to ceftriaxone is cefotaxime (2 g IV every 6 hours). Cefepime (2 g IV every 8 hours), ceftazidime (2 g IV every 8 hours), or meropenem (2 g IV every 8 hours) is preferable when *Pseudomonas aeruginosa* is considered a possible or likely pathogen, such as in patients in the intensive care unit; in the presence of an invasive device; in patients with bedridden status,

prior use of antibiotics, or diabetes mellitus; and in patients who have undergone surgery. (See "Epidemiology, microbiology, and pathogenesis of Pseudomonas aeruginosa infection", section on 'Epidemiology'.)

Patients with penicillin allergies can usually still use one of these agents, potentially with a test-dose procedure (algorithm 1). For patients who have an allergy that precludes the use of beta-lactams and carbapenems (eg, a severe delayed allergy like Stevens-Johnson syndrome), moxifloxacin 400 mg IV daily can be used in addition to vancomycin.

Some experts prefer to give vancomycin **plus** either nafcillin or oxacillin **plus** one of the beta-lactams directed at gram-negative bacteria listed above, while isolation and susceptibility testing of the causative agent is underway. The rationale for this approach is that nafcillin and oxacillin are regarded as the optimal agents for methicillin-susceptible *S. aureus* (MSSA). However, there is not strong evidence to support this, and we generally favor the simpler approach suggested above. (See "Clinical approach to Staphylococcus aureus bacteremia in adults", section on 'Empiric antibiotic therapy'.)

Antibiotics should be directed against the known pathogen if the culture and/or Gram stain of the aspirate is positive. If an aspirate cannot be obtained, the empiric regimen should be continued. (See 'Targeted therapy' below.)

There are no randomized controlled trials evaluating antibiotic regimens for the treatment of SEA. Our approach to antibiotic selection is based on the understanding of the microbiology.

Targeted therapy — Once culture identifies the infecting organism(s), treatment regimens should be simplified and directed to that pathogen.

Antibiotic selection for epidural abscesses are generally similar to that for bacterial meningitis, although epidural abscesses require a longer duration of therapy. Specific regimens for bacterial meningitis are discussed separately. (See "Treatment of bacterial meningitis caused by specific pathogens in adults".)

Given substantial rates of MRSA infections in SEA, vancomycin should be used as initial therapy [50,51]. If *S. aureus* is recovered in culture and susceptibility testing reveals MSSA, therapy should be changed to nafcillin (2 g IV every four hours) or oxacillin (2 g IV every four hours). If the organism is methicillin resistant, vancomycin should be continued and dosed to achieve serum trough concentrations of 15 to 20 mcg/mL. A significant drawback to vancomycin is its poor penetration into the cerebrospinal fluid of approximately 1 and 5 percent in uninflamed and inflamed meninges, respectively [51-54].

Based upon case reports and case series of patients with central nervous system (CNS) infections caused by MRSA, alternatives to vancomycin include linezolid (600 mg IV or orally twice daily) [55-58], trimethoprim-sulfamethoxazole (5 mg/kg of the trimethoprim component IV every 8 to 12 hours) [51,59,60], and daptomycin (6 to 10 mg/kg IV once daily) [61,62]. Although there are insufficient data regarding the efficacy of these regimens for the treatment of CNS infections caused by MRSA, these agents are reasonable alternatives when vancomycin cannot be used or is ineffective. Further studies are needed to establish the benefit of these agents for the treatment of CNS infections. This is discussed in greater detail separately. (See "Treatment of bacterial meningitis caused by specific pathogens in adults", section on 'Staphylococcus aureus'.)

Duration — The appropriate duration of antimicrobial therapy should be determined on a case-by-case basis, taking into account the clinical, laboratory (white blood cell count, erythrocyte sedimentation rate [ESR], CRP), and radiographic response to therapy. The usual duration of antimicrobial therapy is four to eight weeks.

We generally treat patients who have undergone surgical drainage and who have responded well to medical and surgical therapy for four to six weeks. In contrast, we generally treat patients with associated osteomyelitis [1], patients in whom surgical drainage did not occur, and patients in whom the response to therapy has been slow for six to eight weeks.

Follow-up — The first follow-up MRI is obtained at about four to six weeks if the patient is improving, or at any time if clinical deterioration occurs. The ESR can be followed to assure decreasing trends indicative of resolving infection.

PROGNOSIS

About 5 to 7 percent of patients with SEA die due to uncontrolled sepsis or other complications [11,22].

Irreversible paraplegia occurs in 4 to 22 percent of patients [2,3,22,63,64]. Neurologic recovery is unlikely if paralysis is present for more than 24 to 48 hours prior to surgery. In other patients, the degree of neurologic recovery after surgery is related to the duration of the neurologic deficit. In the population of emergency department (ED) patients described above, 23 of 63 patients (37 percent) with a SEA had residual motor weakness [28]. A delay to diagnosis (defined as multiple ED visits before diagnosis, admission without a diagnosis, or more than a 24-hour delay in performance of a definitive study) was associated with an increased risk of residual weakness compared with no diagnostic delay (45 versus 13 percent).

There are limited data on the long-term post-treatment outcomes of patients with neurologic deficits due to SEA. One report of the long-term outcomes of 29 patients admitted to a tertiary care spine center with neurologic deficits due to SEA suggested that some patients show marked to moderate delayed improvement in their motor function following completion of rehabilitation treatment [65].

SUMMARY AND RECOMMENDATIONS

- Spinal epidural abscess (SEA) is a rare but important suppurative infection of the central nervous system. Abscesses that are enclosed within the bony confines of the spinal column can expand to compress the spinal cord and cause severe symptoms, permanent complications, or even death. Prompt diagnosis and proper treatment can avert complications and achieve cure in many cases. (See 'Introduction' above.)

- Bacteria can gain access to the epidural space hematogenously, by direct extension from infected contiguous tissue, or via direct inoculation into the spinal canal. The most common sources are soft tissue infections and complications of spinal surgery or other invasive procedures, including indwelling epidural catheters. However, approximately one-third of patients with SEA have no identifiable source for the infection (table 1). (See 'Pathogenesis' above.)

- *Staphylococcus aureus* accounts for about two-thirds of SEA cases caused by pyogenic bacteria. Methicillin resistance rates among *S. aureus* isolates from SEA have ranged from 25 to 68 percent. (See 'Microbiology' above.)

- The classic diagnostic triad for SEA consists of fever, back pain, and neurologic deficits. However, only a small proportion of patients have all three components at presentation. The initial manifestations are often nonspecific, with fever and malaise. (See 'Clinical manifestations' above.)

- The diagnosis of SEA should be suspected in febrile patients with spinal pain accompanied by an examination consistent with a radiculopathy or other focal neurologic findings, especially if the pain is worsened by palpation or percussion. In patients who have only fever and back pain or only new-onset back pain, a low threshold for suspicion of SEA is warranted if risk factors are present (eg, injection drug use, chronic indwelling venous catheter, distant infected foci, older age, or recent spinal manipulation). (See 'Clinical suspicion' above.)

- Spine imaging should be performed in all patients clinically suspected of having a SEA; magnetic resonance imaging (MRI) with contrast (gadolinium) is the preferred test. In a patient with compatible clinical findings, identification of an epidural collection consistent with abscess is sufficient to make the presumptive diagnosis. (See 'Imaging to confirm diagnosis' above.)

- Once an intraspinal mass that could be an SEA has been identified, it is important to try to isolate the etiologic organism from samples of blood and abscess contents. Two sets of blood cultures should be drawn in all patients with suspected SEA, and either aspirate or surgical specimens of the abscess contents should be sent for cultures. (See 'Identifying the pathogen' above.)

- All patients should have urgent surgical consultation. Surgical decompression and drainage, in addition to systemic antibiotic therapy, is the treatment of choice for many patients and should be performed as early as possible. Surgical intervention is particularly important for those with acute or progressive neurologic deficits, spinal instability, ring-enhancing lesions on MRI, or disease progression on antibiotic therapy. (See 'Surgical decompression for most patients' above.)

- In highly selected patients who do not have risks factors for poor outcomes, in whom aspirates have identified the pathogen and who have no neurologic deficits, conservative medical only approach with antibiotics can be appropriate .(See 'Deciding on surgical versus medical treatment' above.)

- Antibiotics are required and should be started as soon as the diagnosis of SEA is strongly suspected and immediately following the collection of two sets of blood cultures. We do not typically delay antibiotics in order to obtain a pretreatment specimen from the SEA (eg, abscess fluid) to confirm the diagnosis. In patients with SEA, we suggest an empiric regimen of vancomycin and a third- or fourth-generation cephalosporin (**Grade 2C**). Once culture identifies the infecting organism(s), treatment regimens should be simplified and directed to that pathogen. (See 'Antimicrobial therapy' above.)

- The usual duration of antimicrobial therapy is four to eight weeks. It should be individualized based on the clinical, laboratory (white blood cell count, erythrocyte sedimentation rate, C-reactive protein), and radiographic response to therapy. (See 'Duration' above.)

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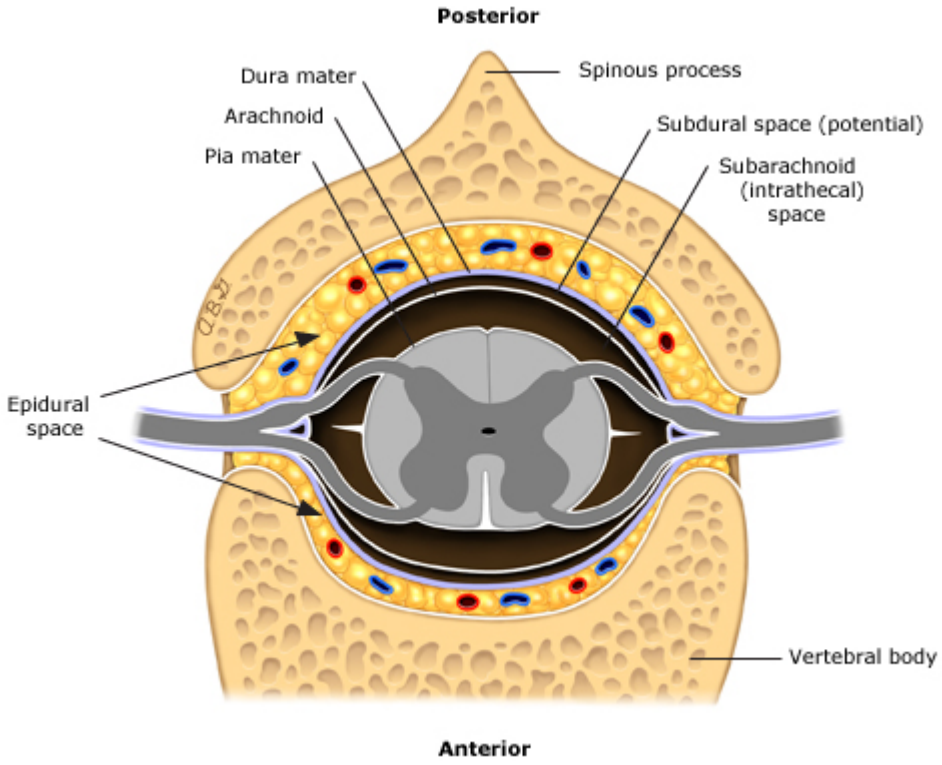
Topic 1292 Version 28.0

GRAPHICS

Transverse section of a vertebral body



Graphic 51884 Version 3.0

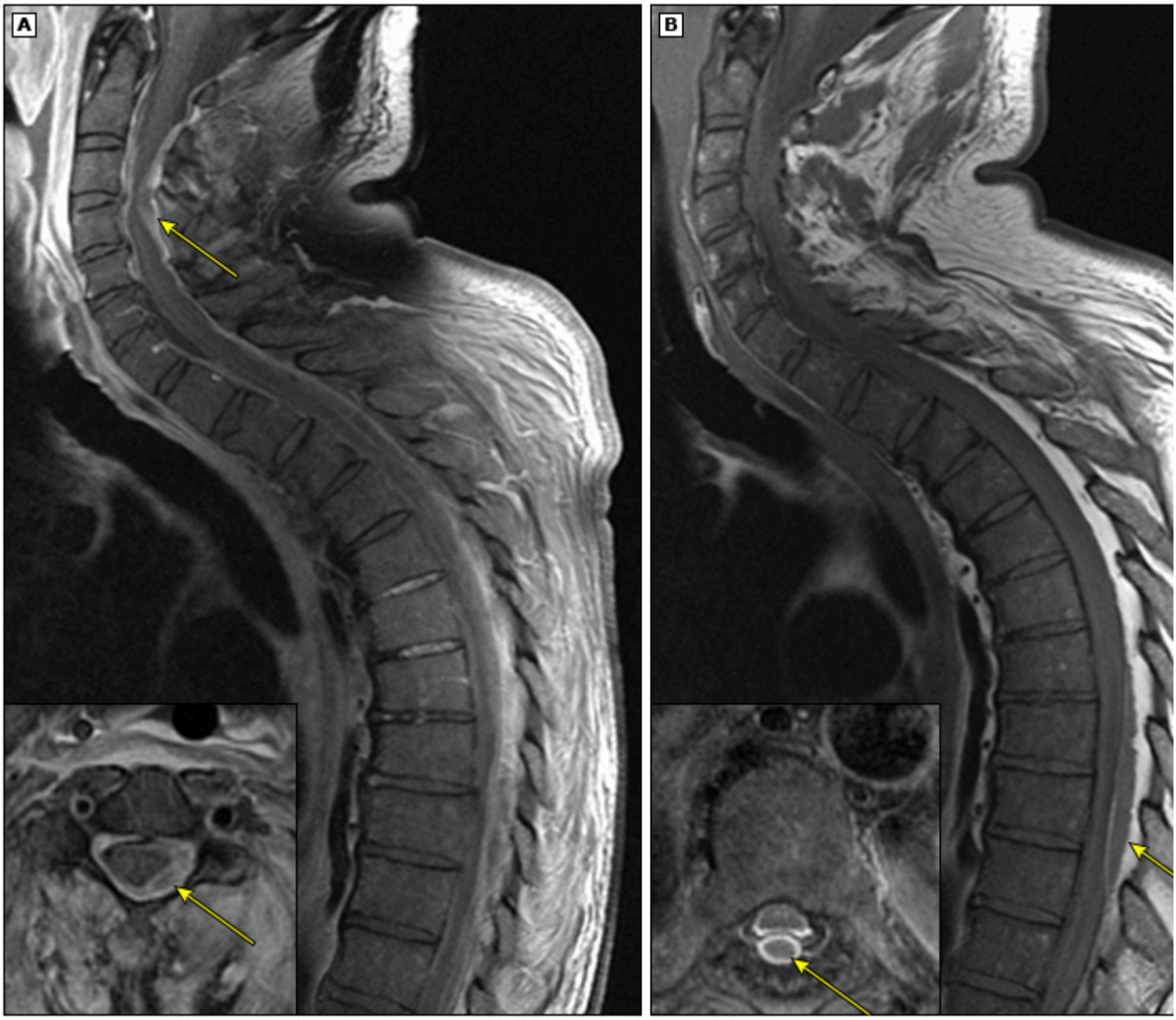
Sources of infection for spinal epidural abscess

Source	Number of patients, percent
No source identified	30
Skin and soft tissue	22
Spinal surgery or procedures	12
Injection drug use	10
Other sources, including epidural catheters	8
Bone or joint	7
Urinary tract	3
Upper respiratory tract	3
Sepsis	2
Abdomen	2
Intravascular catheter associated	<1

Data from Danner R, Hartman BJ. Rev Infect Dis 1975; 9:265; Nussbaum ES, Rigamonti D, Standiford H, et al. Surg Neurol 1992; 38:225; Gellin BG, Weingarten K, Gamache FW Jr, et al. Epidural Abscess. In: Infections of the Central Nervous System, 2nd ed, Scheld WM, Whitley RJ, Durack DT (Eds), Lippincott-Raven Publishers, Philadelphia 1997. p.507; Rigamonti D, Liem L, Wolf AL, et al. Mt Sinai J Med 1994; 61:357; Kapeller P, Fazekas F, Krametter D, et al. Eur Neurol 1997; 38:94.

Graphic 51340 Version 2.0

Spinal epidural abscess

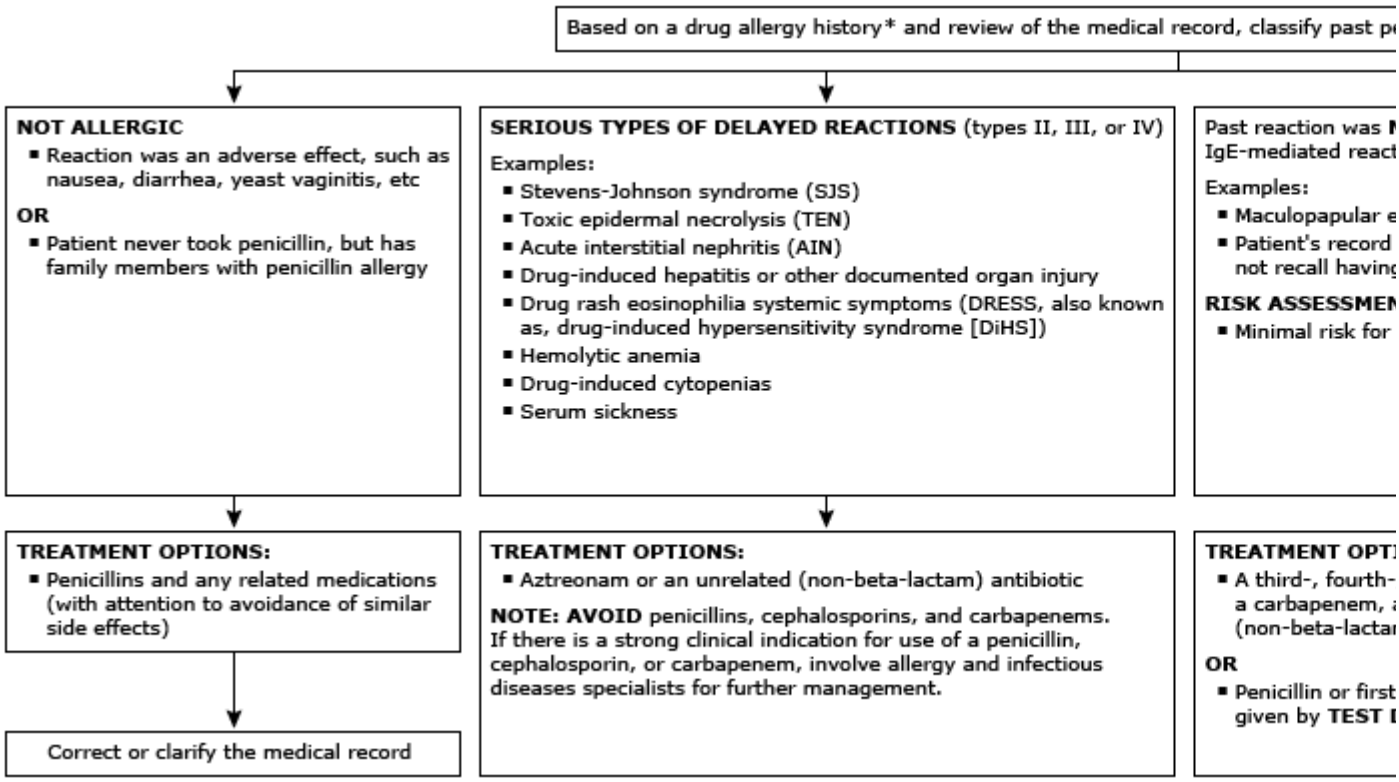


Spinal epidural abscess in a 73-year-old patient. T1-weighted MRI shows epidural lesions (arrow) with surrounding contrast enhancement located at (A) C3/C4 and (B) T7–T9. Insets show corresponding axial sections. *Staphylococcus aureus* was identified in blood and cerebrospinal fluid.

MRI: magnetic resonance imaging.

Graphic 68878 Version 6.0

Approach to the patient with a past penicillin reaction who requires antibiotics



This algorithm is intended for use in conjunction with the UpToDate content on choice of antibiotics in penicillin-allergic hospitalized patients. It is oriented toward hospitalized patients but also applies to outpatients if test dose procedures can be performed in an appropriately monitored setting with the staff and equipment needed to manage allergic reactions, including anaphylaxis.

IgE: immunoglobulin E.

* Ask the following:

1. What exactly were the symptoms?
    - Raised, red, itchy spots with each lesion lasting less than 24 hours (hives/urticaria)?
    - Swelling of the mouth, eyes, lips, or tongue (angioedema)?
    - Blisters or ulcers involving the lips, mouth, eyes, urethra, vagina, or peeling skin (seen in SJS, TEN, other severe type IV reactions)?
    - Respiratory or hemodynamic changes (anaphylaxis)?
    - Joint pains (seen in serum sickness)?
    - Did the reaction involve organs like the kidneys, lungs, or liver (seen in DRESS, other severe type IV reactions)?
2. What was the timing of the reaction after taking penicillin: Minutes, hours, or days later? Was it after the first dose or after multiple doses?
3. How long ago did the reaction happen? (After 10 years of avoidance, only 20% of patients with IgE-mediated penicillin allergy will still be allergic).
4. How was the reaction treated? Was there a need for urgent care or was adrenaline/epinephrine administered?

5. Has the patient tolerated similar medications, such as ampicillin, amoxicillin, or cephalexin since the penicillin reaction?

¶ Isolated mild hives, without other symptoms of an IgE-mediated reaction, can often occur in the setting of an infection. Patients with this history, especially if it occurred in childhood or >10 years ago, may also be considered to be at minimal risk for a recurrent serious reaction.

Δ This algorithm is intended for use in conjunction with additional UpToDate content. For a description of how to safely perform a TEST DOSE PROCEDURE, refer to the UpToDate topic on choice of antibiotics in penicillin-allergic hospitalized patients.

◇ Consult allergist to perform skin testing. If skin testing is not possible, patient may still be able to receive penicillins or first- or second-generation cephalosporins using a desensitization (also known as tolerance induction) procedure. Refer to the UpToDate topic on rapid drug desensitization for immediate hypersensitivity reactions.

Original figure modified for this publication. Blumenthal KG, Shenoy ES, Varughese CA, et al. Impact of a clinical guideline for prescribing antibiotics to inpatients reporting penicillin or cephalosporin allergy. Ann Allergy Asthma Immunol 2015; 115:294.

Graphic 112936 Version 5.0