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REVIEW ARTICLE
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CURRENT CONCEPTS

Spinal Epidural Abscess

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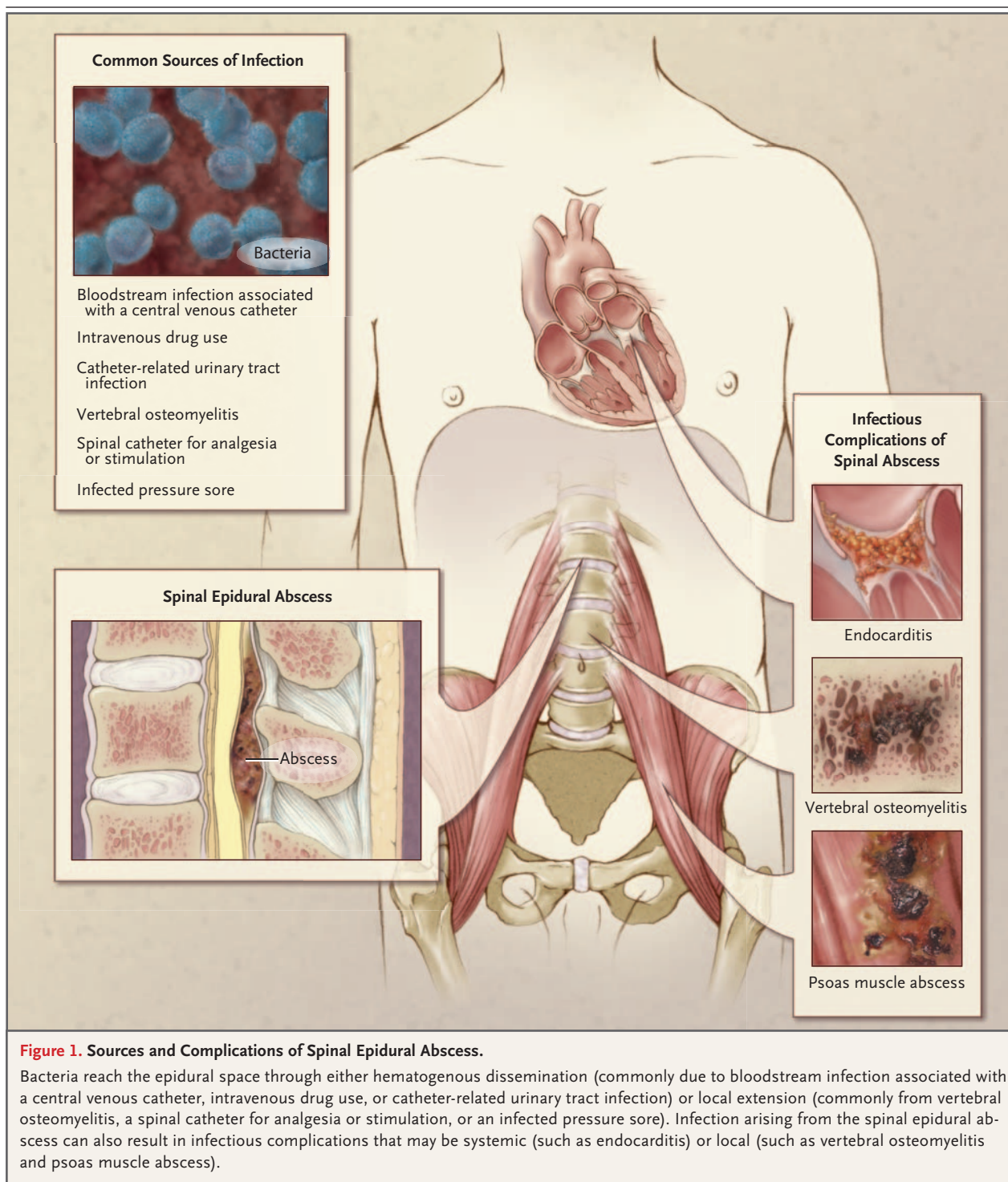
DESPITE ADVANCES IN MEDICAL KNOWLEDGE, IMAGING TECHNIQUES, AND surgical interventions, spinal epidural abscess remains a challenging problem that often eludes diagnosis and receives suboptimal treatment. The incidence of this disease — two decades ago diagnosed in approximately 1 of 20,000 hospital admissions[1] — has doubled in the past two decades, owing to an aging population, increasing use of spinal instrumentation and vascular access, and the spread of injection-drug use.[2-5] Still, spinal epidural abscess remains rare: the medical literature contains only 24 reported series of at least 20 cases each.[1-24] This review addresses the pathogenesis, clinical features, diagnosis, treatment, common diagnostic and therapeutic pitfalls, and outcome of bacterial spinal epidural abscess.

PATHOGENESIS

Most patients with spinal epidural abscess have one or more predisposing conditions, such as an underlying disease (diabetes mellitus, alcoholism, or infection with human immunodeficiency virus), a spinal abnormality or intervention (degenerative joint disease, trauma, surgery, drug injection, or placement of stimulators or catheters), or a potential local or systemic source of infection (skin and soft-tissue infections, osteomyelitis, urinary tract infection, sepsis, indwelling vascular access, intravenous drug use, nerve acupuncture, tattooing, epidural analgesia, or nerve block).[2,5,9,14,16,20,25-33] Bacteria gain access to the epidural space through contiguous spread (about one third of cases) or hematogenous dissemination (about half of cases); in the remaining cases the source of infection is not identified. Likewise, infection that originates in the spinal epidural space can extend locally or through the bloodstream to other sites (Fig. 1). Because most predisposing conditions allow for invasion by skin flora, *Staphylococcus aureus* causes about two thirds of cases.[4,11,25] Although methicillin-resistant *S. aureus* (MRSA) accounted for only 15% of staphylococcal spinal epidural infections just a decade ago,[4] the proportion of abscesses caused by MRSA has since escalated rapidly (up to almost 40% at my institution). The risk of MRSA infection is particularly high in patients with implantable spinal or vascular devices. In these patients abscess may develop within a few weeks after spinal injection or surgery. Less common causative pathogens include coagulase-negative staphylococci, such as *S. epidermidis* (typically in association with spinal procedures, including placement of catheters for analgesia, glucocorticoid injections, or surgery) and gram-negative bacteria, particularly *Escherichia coli* (usually subsequent to urinary tract infection) and *Pseudomonas aeruginosa* (especially in injection-drug users).[2,16,19,22,25,31] Spinal epidural abscess is rarely caused by anaerobic bacteria,[34] agents of actinomycosis or nocardiosis,[25] mycobacteria (both tuberculous and nontuberculous),[2,15,19,25] fungi (including candida, sporothrix, and aspergillus species),[11,19,20,25] or parasites (echinococcus and dracunculus).[25]

Epidural infection can injure the spinal cord either directly by mechanical com-

Figure 1. Sources and Complications of Spinal Epidural Abscess.

Bacteria reach the epidural space through either hematogenous dissemination (commonly due to bloodstream infection associated with a central venous catheter, intravenous drug use, or catheter-related urinary tract infection) or local extension (commonly from vertebral osteomyelitis, a spinal catheter for analgesia or stimulation, or an infected pressure sore). Infection arising from the spinal epidural abscess can also result in infectious complications that may be systemic (such as endocarditis) or local (such as vertebral osteomyelitis and psoas muscle abscess).

pression or indirectly as a result of vascular occlusion caused by septic thrombophlebitis. Histopathological features in patients with spinal epidural abscess are illustrated in Figure 2. Although experiments in rabbits have revealed a primary role for mechanical compression,[35] other animal models show that compression and ischemia have an additive adverse effect on neurologic function. The remarkable degree of neurologic improvement in some patients after decompressive
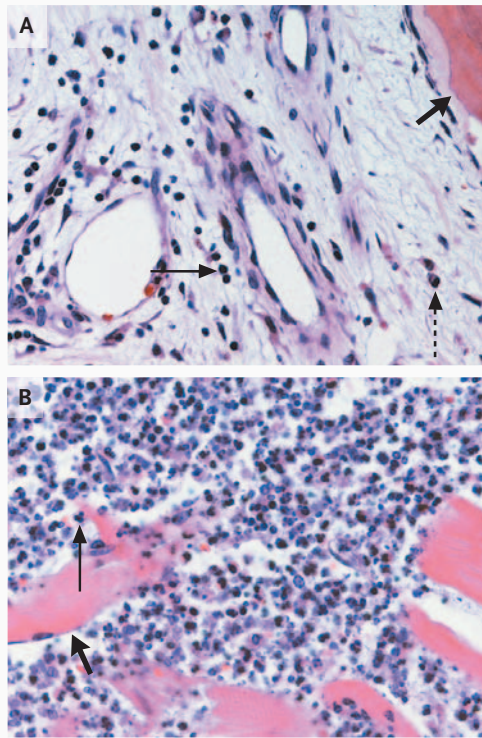
Figure 2. Histopathological Findings in Patients with Spinal Epidural Abscess.

Panel A, which delineates the histopathological findings of vertebral-bone biopsy in a patient with a history of chronic vertebral osteomyelitis that led to spinal epidural abscess, shows chronic inflammatory cells, including lymphocytes (arrow) and plasma cells (dashed thin arrow) in the vicinity of trabecular bone (thick arrow) (hematoxylin and eosin). Panel B provides a histopathological illustration of how subsequent contiguous spread of infection from the epidural space resulted in the formation of an acute psoas muscle abscess, which is demonstrated by the presence of infiltrates of polymorphonuclear cells (thin arrow) adjacent to striated muscle fibers (thick arrow) (hematoxylin and eosin).
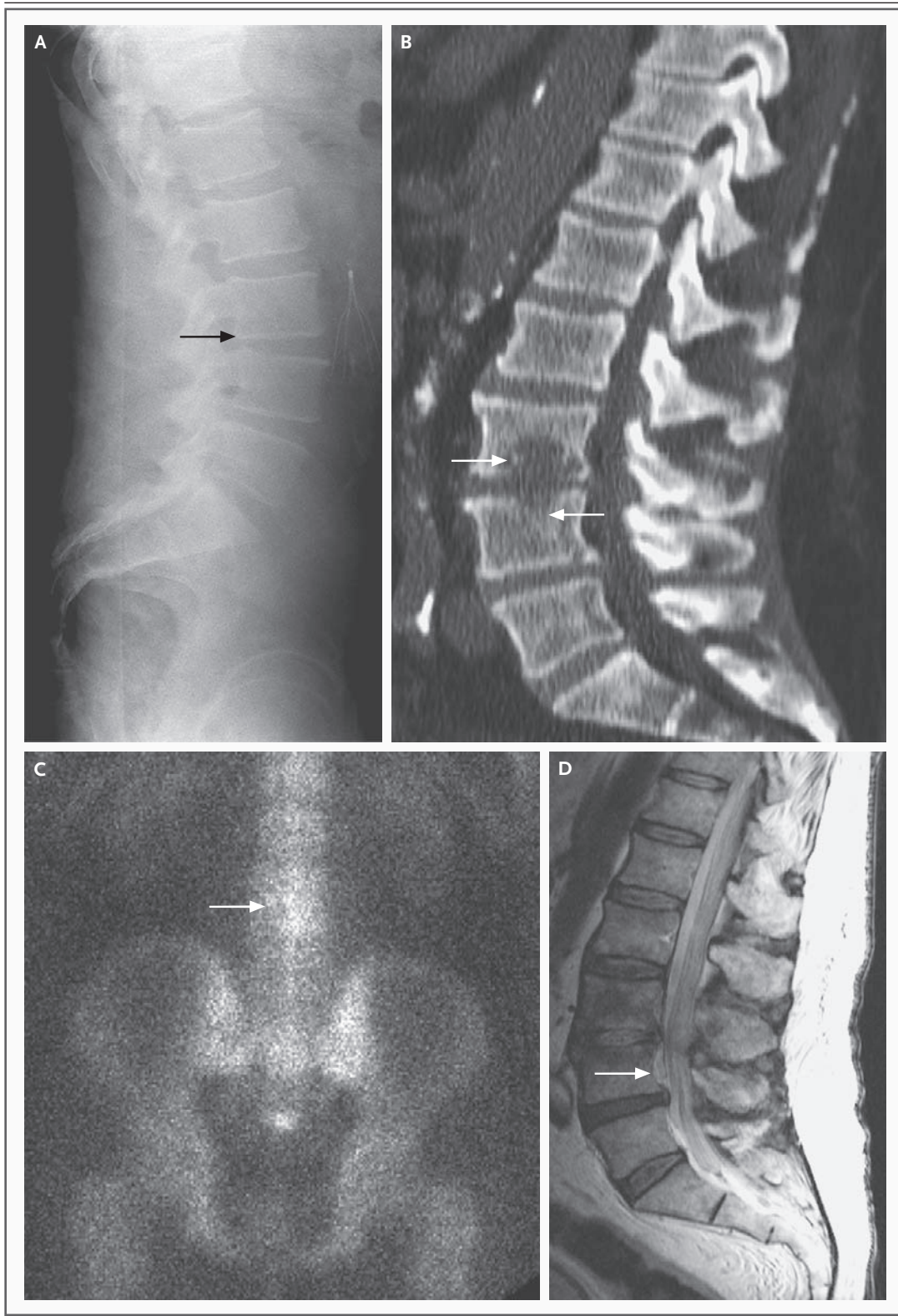
laminectomy provides evidence of a mechanical pathophysiology, but thrombosed levels are observed in few postmortem examinations.[36] Furthermore, infarction of the spinal cord, as reflected by altered cord signal on magnetic resonance imaging (MRI), can be caused not only by vascular occlusion stemming from septic thrombophlebitis but also by profound compression. Therefore, the principal mechanism of cord damage caused by spinal epidural abscess remains uncertain and may differ among patients.

CLINICAL FEATURES

An established staging system outlines the progression of symptoms and physical findings: stage 1, back pain at the level of the affected spine; stage 2, nerve-root pain radiating from the involved spinal area; stage 3, motor weakness, sensory deficit, and bladder and bowel dysfunction; and stage 4, paralysis. Although patients with stage 2 cervical or lumbar abscess usually have neck pain radiating to the arms or low back pain radiating down the legs, respectively, the clinical presentation of stage 2 thoracic abscess with chest or abdominal pain can be more enigmatic,[37] particularly in patients who have other, more common reasons for such symptoms. Back pain (present in about three quarters of patients), fever (documented in almost half of patients), and neurologic deficit (detected in about one third of patients) are the three most common symptoms.[3,4,25] However, this classic clinical triad of back pain, fever, and neurologic deficit is present only in a minority of patients.[9] Both the duration of the symptoms before hospital admission (range, 1 day to 2 months) and the rate of progression from one stage to another (neurologic deficit and eventual paraplegia can evolve in a matter of hours to days) are highly variable.[1] Because abscesses are more likely to develop in larger epidural spaces that contain infection-prone fat, they are more common in posterior than anterior areas and in thoracolumbar than cervical areas.[1,3,21] The escalating use of spinal interventions for pain management has led to a disproportionate increase in the occurrence of lumbar epidural infection.[11] Spinal epidural abscesses generally extend over three to four verte-

brae,[1,14,15,17,20,21] but in rare cases they involve the whole spine, resulting in so-called panspinal infection.[4,38]

DIAGNOSIS

A diagnosis of spinal epidural abscess is suspected on the basis of clinical findings and supported by laboratory data and imaging studies but can be confirmed only by drainage. Although leukocytosis is detected in about two thirds of patients[1,16] and inflammatory markers (erythrocyte sedimentation rate and C-reactive protein) are almost uniformly elevated,[5,14,16,20] neither the presence nor the degree of these laboratory abnormalities is specific for spinal epidural abscess. Bacteremia causing or arising from spinal epidural abscess is detected in about 60% of patients,[7,21] more so in those infected with *S. aureus* than with other organisms.[1,7,20,21] The presence of *S. aureus* bacteremia does not, in and of itself, establish the source of infection because *S. aureus* is also the cause of a number of mimicking conditions, such as osteomyelitis, diskitis, sepsis, and endocarditis. In three quarters of patients whose cerebrospinal fluid (CSF) is evaluated, CSF analysis shows a high level of protein and pleocytosis (with either a polymorphonuclear or a mononuclear predominance), findings that are suggestive of parameningeal inflammation but are not specific for epidural infection.[1] The results of Gram staining of CSF is usually negative, and CSF cultures are positive in less than 25% of patients whose CSF is microbiologically assessed. However, blood cultures yield the infecting pathogen in almost all patients with a positive CSF culture.[1] Furthermore, although rare, there is a risk of meningitis or subdural infection if the needle traverses the epidural abscess and of neurologic deterioration if the lumbar puncture is performed below a complete spinal subarachnoid block.[1,20,21,39] Because lumbar puncture affords meager information and is associated with a slight potential risk, it should not be done routinely; CSF should be analyzed only if myelography is performed.

Both MRI with intravenous administration of gadolinium and myelography followed by computed tomography (CT) of the spine are highly sensitive (more than 90%) in diagnosing spinal epidural abscess.[4,20] However, MRI is the imaging method of choice because it is less invasive, delin-

eates both the longitudinal and paraspinal extension of the abscess (which is essential for planning surgery), and may help differentiate infection from cancer on the basis of the appearance and the signal intensity of the image.[40] A plain roentgenograph or CT of the spine may reveal narrowing of the disk and bone lysis to indicate the presence of diskitis and osteomyelitis (which coexist with spinal epidural abscess in up to 80% of patients),[11] and radionuclide scanning (with technetium, gallium, or indium) may show increased uptake, helping to identify the affected site. However, the findings of these imaging tests are neither sensitive nor specific for spinal epidural abscess and should not take the place of MRI (Fig. 3).

Because spinal epidural abscess is a rare condition that may appear with nonspecific findings of back pain, fever, leukocytosis, or a high erythrocyte sedimentation rate or C-reactive protein level, it is often misdiagnosed on presentation, particularly in neurologically intact patients (those in stage 1 or stage 2).[1,9,14] Instead, more common infectious conditions (osteomyelitis, diskitis, meningitis, urinary tract infection, sepsis, and endocarditis) and noninfectious conditions (intervertebral-disk prolapse, degenerative joint disease, spinal tumor, demyelinating illness, transverse myelitis, and spinal hematoma) are frequently diagnosed at the time of initial evaluation.[1,20,25]

TREATMENT

Owing to the rare occurrence and serious outcome of spinal epidural infection, it is both impractical and ethically difficult to conduct prospective, randomized clinical trials to determine the optimal treatment. However, the majority of retrospective studies provide support for the overwhelming consensus that surgical drainage together with systemic antibiotics is the treatment of choice.[1,2,4,5,7,15,20,21,23,24] Because the preoperative neurologic stage is the most important predictor of the final neurologic outcome, and because the rate of progression of neurologic impairment is difficult to predict (with some patients becoming paralyzed within hours after the onset of neurologic deficit), decompressive laminectomy and débridement of infected tissues should be done as soon as possible.[1,3,9,15]

A few retrospective studies have reported similar outcomes in patients who were treated with antibiotics alone and in patients who received

both medical and surgical treatment.[6-8,10] However, in these studies, the group of patients receiving antibiotics alone had no or minimal neurologic impairment[6,7,10] or smaller abscesses,[8] and in some of these patients, neurologic deterioration occurred despite the use of appropriate antibiotics.[1,7,18,20,21] The true index of the success of nonsurgical therapy is difficult to discern both because cases may have been selectively reported[18] and because unsuccessful attempts at conservative management are rarely reported once a decompressive laminectomy is performed.[41]

Figure 4 shows an algorithm for treating patients with diagnosed spinal epidural abscess. In clinical scenarios in which decompressive laminectomy is declined by the patient, contraindicated because of high operative risk, unlikely to reverse paralysis that has existed for more than 24[1,4] to 36[21,24] hours, or considered impractical because of panspinal infection, patients may be treated medically. Patients who are neurologically intact may also qualify for nonsurgical therapy if the microbial cause is identified and the patients' clinical condition is closely monitored. Although controversial, this approach may be reasonable especially when the radiologic epidural abnormality and the symptoms can be explained by finding (including postoperative changes) that the inflammation is not caused by a true abscess. Antibiotic therapy must be guided by the results of blood cultures or a CT-guided needle aspiration of the abscess.[42,43] Although emergency decompressive laminectomy is not indicated in patients with paralysis that lasts longer than 24 to 36 hours, this surgery may still be needed to treat the epidural infection and control sepsis. Because it is impractical to perform decompressive laminectomy along the whole spine in patients with panspinal epidural abscess (Fig. 5), the physician may want to consider less extensive surgery, such as a limited laminectomy or laminotomy with cranial and caudal insertion of epidural catheters for drainage and irrigation.[39]

Pending the results of cultures, empirical antibiotic therapy should provide coverage against staphylococci (usually with vancomycin to cover MRSA) and, because of the potentially serious consequences, gram-negative bacilli (potentially with a third- or a fourth-generation cephalosporin, such as ceftazidime or cefepime, respectively), particularly in the presence of documented or suspected gram-negative bacterial infection of
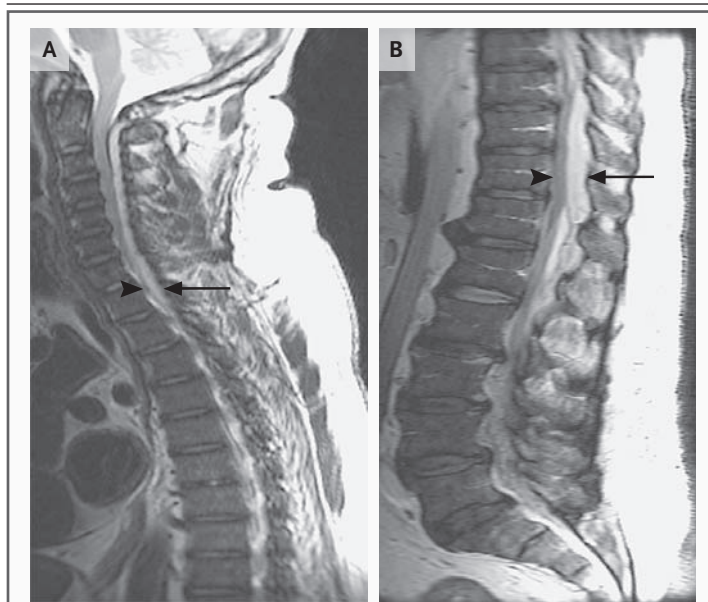

Figure 4. Management of Spinal Epidural Abscess.
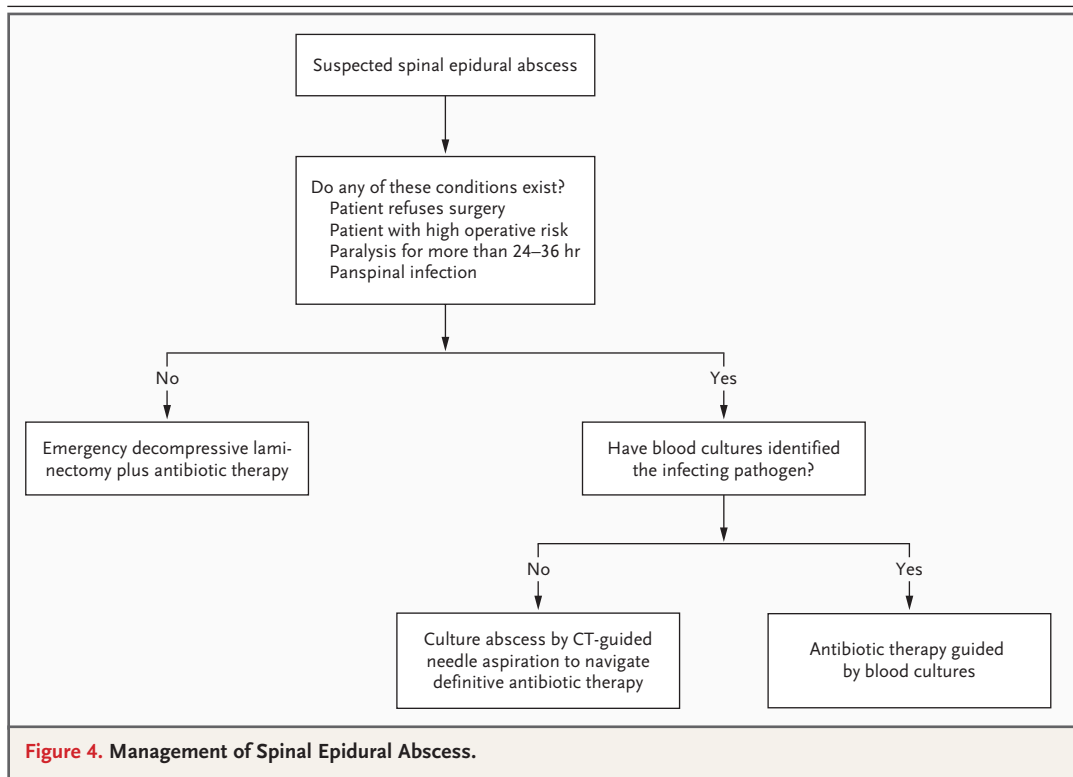
Figure 5. Imaging Findings in a Patient with a Panspinal Epidural Abscess.
In Panel A, MRI shows a posterior collection of epidural fluid (arrow at C7) that extends from C1 to T8 and displaces the ventrally located thecal sac (arrowhead at C7) in a patient in whom quadriplegia developed as a result of infection with MSSA. In Panel B, MRI of the remaining spinal column from T8 to the lumbosacral region demonstrates the caudal extension of the same posterior spinal epidural abscess (arrow at T11) and anterior displacement of the spinal cord (arrowhead at T11).

other sites, such as the urinary tract. Because vancomycin is less active than β-lactam agents against methicillin-susceptible *S. aureus* (MSSA), nafcillin or cefazolin is preferred for treatment of documented MSSA infection. The usual duration of antibiotic therapy is at least 6 weeks because vertebral osteomyelitis exists in most patients with spinal epidural abscess. Because noncompliance and limited bioavailability may impede the effectiveness of oral therapy, intravenous administration of antibiotics is preferred.

Neurologic function, signs of sepsis, and imaging findings should be closely monitored after treatment begins, particularly in patients who are treated medically. Subsequent development of an immunocompromising condition or intake of immunosuppressive agents may result in recurrence of spinal epidural abscess long after the completion of antibiotic therapy.[41] In patients with spinal epidural abscess associated with an infected spinal cord stimulator, it is crucial to remove the whole stimulator system (including the subcutaneously placed generator and epidural electrodes) to reduce the likelihood of recurring implant-

related epidural infection.[44] Patients with unexplained persistent or recurrent epidural infection may be assessed for rare sources of infection, such as esophageal tear (in the case of cervical epidural abscess) or intestinal–spinal fistula (in the case of thoracolumbar abscess). Although there have been sporadic reports in which glucocorticoid therapy has been associated with an adverse outcome in patients who already had a severe case of spinal epidural abscess,[21] it may help to reduce swelling in patients with progressive neurologic compromise who are awaiting surgical decompression.

DIAGNOSTIC AND THERAPEUTIC PITFALLS

Irreversible paralysis, the most fearsome complication of spinal epidural abscess, continues to affect 4 to 22% of patients.[1,17,21,23] Although bacterial virulence and host characteristics may contribute to a poor outcome, delayed diagnosis and suboptimal management are the usual culprits. Overall, about half (range, 11 to 75%)[9,14] of cases are initially misdiagnosed; fortunately, not all diagnostic delays are associated with deterioration in neurologic function and worsening of sepsis. Although promptly diagnosed cases may still be improperly treated, suboptimal management often follows a delay in diagnosis. Table 1 summarizes common diagnostic and therapeutic pitfalls and ways to avoid their potential serious sequelae.

OUTCOME

The single most important predictor of the final neurologic outcome is the patient's neurologic status immediately before surgery.[1,15,20,21,23,24] Unless perioperative complications occur, the final neurologic condition in patients in whom the spinal epidural abscess is adequately decompressed is as good as or better than the preoperative condition. Patients who undergo surgery during stage 1 or stage 2 are expected to remain neurologically intact and possibly have a decreased risk of back and radicular pain, and those in stage 3 may have no weakness or a lesser degree of weakness after surgery than before surgery. Patients in stage 4 who have been paralyzed for up to 24 to 36 hours are likely to regain some neurologic function postoperatively. Not unexpectedly, there are no published data comparing the postoperative outcome in patients who have been paralyzed for

Table 1. Common Diagnostic and Therapeutic Pitfalls and Recommended Approaches.

Pitfall	Recommendation
Ordering imaging studies of an area that is not the site of epidural infection	Clinically assess patients for spinal tenderness and level of neurologic deficit to more accurately identify the region to be imaged.
Identifying only one of multiple nonadjacent epidural abscesses	Suspect the presence of other undrained abscesses if bacteremia persists or neurologic level changes after surgery.
Ascribing all clinical and laboratory findings to vertebral osteomyelitis	Determine whether osteomyelitis is associated with epidural abscess, particularly if a neurologic deficit is evident.
Being unable to adequately evaluate sensorimotor function in patients with altered mental status	Check for depressed reflexes and bladder or bowel dysfunction, which can indicate spinal cord injury.
Asking nonphysicians who may not appreciate the urgency of the case to order consultations for patients with suspected or documented epidural abscess	Directly communicate with consultants to ensure timely diagnosis and treatment.
Surgically managing a spinal stimulator–associated epidural abscess by removing only the implant	Decompress the abscess to preserve neurologic function and remove the implant to increase the likelihood of curing the infection.
Medically treating *S. aureus* bacteremia without attempting to identify the source	Consider a spinal source of infection if clinically indicated.

various periods during the surgical window of opportunity of 24 to 36 hours. Earlier surgery in some patients with virulent infection and rapid deterioration in their neurologic condition may be associated with a better outcome. Likewise, a neurologic deterioration between admission and accurate diagnosis may lead to a poorer outcome.[1,21] Although MRI findings (related to the length of the abscess and the extent of spinal-canal stenosis),[45] degree of leukocytosis,[16] and level of elevation of the erythrocyte sedimentation rate[15] or C-reactive protein[16] were reported to correlate with outcome, these potential relationships were identified by univariate analyses that did not consider the pretreatment neurologic status and, therefore, need to be further investigated.

About 5% of patients with spinal epidural abscess die, usually because of uncontrolled sepsis, evolution of meningitis, or other underlying illnesses. The final neurologic outcome and functional capacity of patients should be assessed at least 1 year after treatment, because until then, patients may continue to regain some neurologic function and benefit from rehabilitation. The most common complications of spinal cord injury are pressure sores, urinary tract infection, deep-vein thrombosis, and in patients with cervical abscess, pneumonia.[16] Optimal outcome requires well-coordinated multidisciplinary care by emergency medicine physicians, hospitalists, internists, infectious-disease physicians, neurologists, neurosurgeons, orthopedic surgeons, nurses, and physical and occupational therapists.

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