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 medical therapy.
antimicrobial therapy alone. 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. 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. The increased recognition of this infection likely relates to the improved accuracy in diagnoses imparted by magnetic resonance imaging (MRI). 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. 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. 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.

**Historic Perspective: Epidural Spine Infections**

Albers is credited with the first report of SEA, and in 1853, Ducek termed this condition “peripachymeningitis,” which by later reports was identified as “pachymeningitis externa.” 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%. In 1948, Heusner delineated the classical clinical features of SEA, which remain valid today.

**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.

At present, numerous proponents recommend urgent surgical decompression as the treatment of choice for SEA. 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. 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 dissemination**

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. 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 ligamentum flavum 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 sacroccygeal 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. 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. 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. Finally, as found in several studies, SEA is predominantly identified in the thoracic and lumbosacral region.

**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. 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. 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. 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.

**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.

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.

**Most common presenting symptoms**

The most common presenting symptoms include back pain (85%), fever (50%), and neurologic deficit (32%). This classic triad of symptoms, however, presents in the minority of patients. 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. 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.

**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. 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. **[20]**
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 than one-quarter of patients.[14]

**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.[20,41,42] 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.[16,26] Sagittal MR of the entire spine is 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             |

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

| Marker                    | Level             |
|---------------------------|-------------------|
| Serology                  | Positive 60% of cases |
| Blood cultures            | Elevated          |
| 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.[22] 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 neurologic symptoms that have been stable for over 72 hours.[49] 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.[21]

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 | Homogenous collection or rim-enhancement only |
| T2-Weighted sequence | Increased | |
| STIR | Increased in the setting of vertebral osteomyelitis |

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

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

**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.

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