Curr Infect Dis Rep (2014) 16:436
DOI 10.1007/s11908-014-0436-7

CENTRAL NERVOUS SYSTEM INFECTIONS (J LYONS, SECTION EDITOR)

Spinal Epidural Abscess

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Published online: 18 September 2014

Abstract Spinal epidural abscess (SEA) remains a relatively infrequent diagnosis. *Staphylococcus aureus* is the most common organism identified, and the infectious source in SEA emanates from skin and soft tissue infections in about 20 % of instances. The thoracic spine is most often involved followed by the lumbar spine. The classic triad of fever, spinal pain, and neurological deficit is present in but a minority of patients. The appearance of neurological deficits with SEA has a significant impact on the prognosis; therefore, early diagnosis is imperative. Magnetic resonance imaging has permitted earlier diagnosis, although significant delays in diagnosis are common due to the nonspecific symptoms that frequently attend the disorder. Due to the rarity of this condition, there have been few randomized controlled trials to evaluate new treatment strategies, and most recommendations regarding treatment are based on case series studies often derived from the experiences at a single center.

Keywords Spinal epidural abscess · Spine · Infection · Magnetic resonance imaging · Myelopathy · Radiculopathy

Introduction

Physicians must remain vigilant for the possibility of spinal epidural abscess (SEA). The presenting manifestations may be nonspecific and subacute, and chronic SEA may be remarkably insidious in nature. Fever is present in about two thirds of patients with SEA, and spinal pain, though present in 90 %, is not invariable. The disorder is attended by high morbidity and mortality if not treated promptly and appropriately. We review the incidence, etiology, pathogenesis, clinical manifestations, diagnostic measures, and treatment of SEA.

Incidence

Although the incidence of spinal epidural abscess (SEA) has been reported to have increased over the past several decades, especially since the late 1980s [1–3], it still remains a relatively rare disorder. A search of PubMed for each of the last 5 years returns no more than 20 papers published annually with the term "spinal epidural abscess" in the title. The incidence rate of SEA reported in 1975 was 0.2–1.2 cases/10,000 [4]. Whereas, in a retrospective study from 1990 that spanned 10 years, SEA accounted for 1.96 of every 10,000 hospital admissions [1]. A number of reasons have been postulated for this increase including the widespread availability of magnetic resonance imaging (MRI), an aging population, and an increase in the prevalence of intravenous drug use (IVDU) among others [2]. Traditionally, SEA has been predominantly considered as a disease of the adult population [5], although pediatric cases are often reported in the literature [6•, 7, 8] with cases reported in infants as young as 10 days [9]. SEA was thought to occur with an equal male-to-female ratio [10], but more recent studies suggest a male preponderance [5]. A number of risk factors have been consistently noted to be associated with the occurrence of spinal epidural abscess including diabetes mellitus, IVDU, renal failure patients on hemodialysis, alcohol abuse, trauma, indwelling vascular devices, HIV, immunocompromised patients and use of immunosuppressant medications, spinal instrumentation, and epidural spinal procedures [1, 2, 5, 11].

This article is part of the Topical Collection on *Central Nervous System Infections*

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Etiology

Bacterial infections are the overwhelmingly most common cause of SEA. *Staphylococcus aureus* (both methicillin sensitive and resistant) is the predominant bacterial etiology [1, 2, 4, 5, 11, 12], but a broad spectrum of other bacteria are also seen including *Streptococcus pneumoniae*, coagulase-negative *Staphylococcus*, and Gram-negative bacilli, such as *Escherichia coli* and *Pseudomonas* [1, 2, 4, 5, 11–16]. Rarer organisms include other bacteria, such as *Bartonella henselae* [17], *Brucella* [18], *Nocardia* [7, 8], anaerobic bacteria [5], tuberculosis [19–21], fungal infections such as aspergillus [22, 23], actinomycetes [24, 25], and even parasites [5, 26]. Polymicrobial infections have also been observed [27, 28]. Spinal epidural abscess due to fungal infection was often observed during the recent epidemic of contaminated corticosteroid provided by the New England Compounding Center following injection therapy [29, 30, 31•, 32, 33].

Pathogenesis

The offending organism gains entry into the epidural space by various mechanisms including hematogenous spread from distant sites, such as skin and soft tissue infections, or by contiguous spread from an adjacent site [5, 34]. A primary source of infection can be identified in about 60 % of cases [10]. Skin and soft tissue infection appears to be the most common source accounting for about one fifth of established sources. Other possible sources reported include bone and joint infections, urosepsis, prostatic abscesses, dental abscesses, deep neck space infections such as retropharyngeal abscesses, endocarditis, thoracic and mediastinal infections, and abdominal sources [4, 5, 10, 16, 24, 35–39].

The exact mechanism by which the epidural abscess causes neurological injury remains controversial to date. The widespread belief is that it is a combination of direct mechanical compression by pus with or without associated vascular damage [1, 5, 40, 41]. The vascular injury may be the consequence of compression of the intrinsic circulation of the spinal cord or, less likely, inflammation of the blood vessels.

The most common site of SEA appears to be in regions where there is excessive epidural pad of fat and the spinal cord is relatively narrow [34], and, consequently, the most common sites include the thoracic and lumbar regions either alone or as part of multilevel disease [1, 5, 11]. Percentages derived from several series indicate that the thoracic spine is involved in about 50 % followed by the lumbar spine at 35 %, and the cervical spine in 14 % [4, 10, 21, 34, 39, 42, 43]. The abscess is posterior in 79 % of cases [4, 10, 34, 39]. Typically, the abscesses involve multiple segments by the time of diagnosis. Most abscesses are posteriorly located since the cord is more adherent anteriorly to the ligaments. Anteriorly located abscesses are typically associated with vertebral osteomyelitis [34, 44].

Clinical Features

Although not universally observed, the classic description by Heusner [34] regarding the four stages of SEA evolution is still valid and helps in understanding the disease process. These stages include stage I characterized by spinal pain; stage II signaled by root pain; stage III in which there is involvement of motor, sensory, and sphincter function; and stage IV in which paralysis has ensued. The classic clinical triad of back pain, fever, and neurological deficits is present only in a small percentage of the patients [45], and initial misdiagnosis is common. In one series, an alternate diagnosis was considered in about one half of all patients with SEA [10]. The most common presenting feature is spinal pain seen in about 90 % followed by neurological deficit (80 %), and fever (67 %) [46]. Neurological symptoms range from radicular to myelopathic features [5, 11]. The duration of symptoms before presentation and the rate of progression of symptoms are both very variable [1, 21]; patients with chronic symptoms from SEA are often diagnostically challenging.

Low back pain is one of the most common presenting complaints in most emergency departments [47]. Only a very small percentage of the patients presenting with low back pain actually have SEA [48], and hence, there is often a significant diagnostic delay with multiple emergency room visits or hospital admission with an incorrect diagnosis frequently preceding an accurate diagnosis [15, 45]. In addition to the classic clinical features of back pain, fever, and neurological symptoms, SEA can often manifest with unrelated presentations such as acute abdomen [49] and meningitis [50], depending on their location. A high index of suspicion is necessary in order to avoid unnecessary delays in diagnosis and management.

Diagnosis

Unless vertebral osteomyelitis or disk space infection is present, plain X-rays of the spine are generally unrevealing. MRI has fundamentally altered the way that SEA is diagnosed. Gadolinium-enhanced MRI (Fig. 1) has replaced CT myelogram as the imaging modality of choice. It has shown very good sensitivity and specificity. SEA appears isointense or hypointense on T1-weighted imaging and hyperintense on T2-weighted imaging with a linear enhancement pattern seen on the post contrast sequences [1, 2, 5, 11, 51, 52]. However, overreliability on MRI to make the diagnosis can also be misleading, as the interpretation of the MRI is not very straightforward and diagnosis can be missed because of

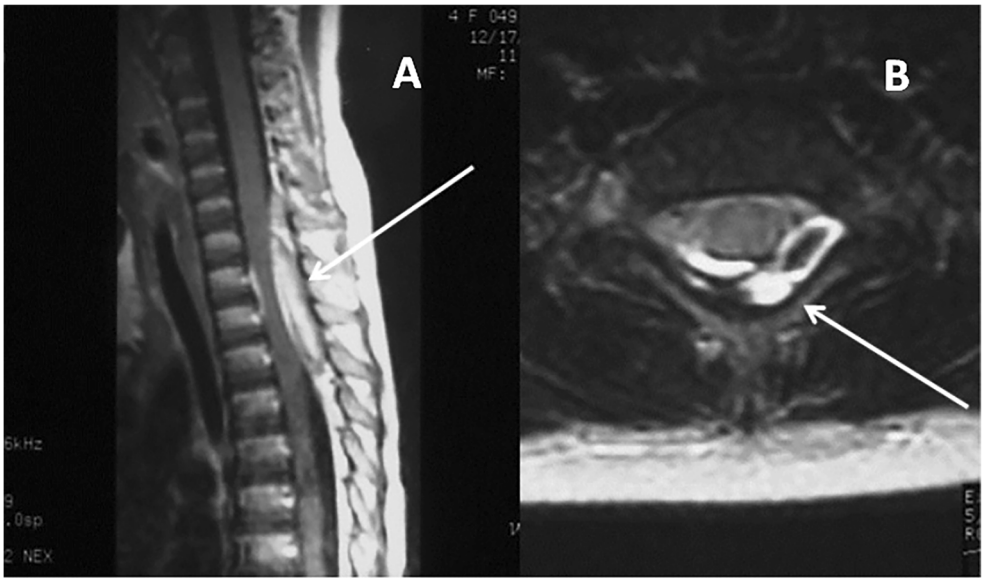

Fig. 1 Thoracic spine magnetic resonance image (sagittal and axial images). T1-weighted MR sagittal imaging (**a**) with gadolinium shows a longitudinally extensive, contrast enhancing lesion (*arrow*) posterior to the spinal cord in the midthoracic region. On axial imaging (**b**), a large contrast-enhancing lesion with loculation is seen posterior to the spinal cord (*arrow*)

patient motion and inexperience of the interpreting radiologist [53]. Other diagnostic measures that have been employed include radionuclide scans with gallium and technetium, but these studies are seldom required and are often misleading [54]. In patients in whom there is a high index of suspicion of SEA who have a negative MRI, CT myelography should be undertaken.

In light of the frequency of low back pain in the general population and the cost and time involved in MR imaging, selecting patients for MRI is important. Several case series have suggested the use of inflammatory markers such as erythrocyte sedimentation rate (ESR), C-reactive protein (CRP), and elevated white cell count as a screening tool [1–3, 46, 55••]. The mean ESR in a meta-analysis of 915 cases was 77 mm (range 1 to 150 mm) and the mean white cell count was 15,700 cells/μL (range, 1,500 to 42,000 cells/μL) [5]. A practical approach proposed includes identifying high risk factors, such as parenteral drug abuse, hemodialysis, immunocompromised, diabetes mellitus, and alcohol abuse, and screening them for inflammatory markers. Any patient with (1) back pain associated with fever or (2) neurological deficits with or without risk factors, back pain, or fever should undergo emergent MRI to rule out the abscess. This diagnostic approach is sensitive and decreases delays in diagnosis [55••].

Treatment

The currently accepted standard of care includes surgical drainage accompanied by prolonged antibiotic coverage for a minimum of 4 weeks. Certain studies challenged the necessity for surgical drainage and demonstrate good outcomes with purely conservative treatment [56, 57]. However, these studies included patients without major neurological deficits. Studies comparing early surgical approach versus conservative treatment showed that conservative treatment was associated with high failure rates requiring eventual surgeries and also risk of poor outcome due to delayed surgery [12, 58•]. Once neurological deficits have developed, the opportunity for a good prognosis diminishes significantly. The earlier the epidural abscess is diagnosed and treated, the better the neurological outcome. Immediate surgical decompression combined with the appropriate antibiotic therapy is critical in managing SEA presenting with a focal neurological deficit [58•].

A number of factors have been proposed as predictors for failure of conservative treatment, the most notable of them include age>65 years, diabetes mellitus, bacteremia especially with MRSA, and neurological deficits [12, 46, 59] Medical management without surgical intervention might be considered in the following instances: (i) absence of neurological deficits, (ii) panspinal involvement making surgery less feasible, (iii) presence of neurological deficits >24–36 h, and (iv) patients who are very poor surgical candidates [2, 34]. The first group of patients needs to be monitored closely and may actually be safer with early surgical intervention.

Percutaneous drainage of the abscesses under CT guidance can be used as an alternative if the lesion is very dorsally located and if the patients are not surgical candidates either due to the extent of the disease or due to comorbid conditions. It has the added advantage of accessing tissue for culture when compared to pure medical management [60, 61].

The most common surgical approach used is laminectomy through a posterior approach and abscess drainage. However, this may vary depending on the location of the abscess. A number of other surgical options have been described including an anterior decompression with debridement and primary grafting or a combined approach [2]. Minimally invasive

surgical techniques appear to be a promising alternative for multilevel abscesses that are not be amenable to open surgery due to concerns of spinal stability [62].

The initial choice of antibiotic should provide broad-spectrum coverage targeting primarily Gram-positive organisms, such as *S. aureus*, and Gram-negatives, such as *E. coli* and *Pseudomonas*. Vancomycin in combination with a third-generation cephalosporin or an aminoglycoside is a reasonable initial empiric choice [2], but antibiotic selection should be tailored to the organism subsequently isolated. Generally, cultures obtained from the abscess site are preferred over blood cultures, although blood cultures have shown a good correlation with abscess cultures [2, 56, 58•]. Culture of pus from the abscess reveals the organism in about 90 % of cases, while blood cultures are positive in 60 % of instances and cerebrospinal fluid in 17 % [10, 21]. The duration of treatment is highly variable, but the currently accepted duration include a minimum of 4 weeks with further duration of treatment being based on clinical, laboratory, and imaging resolution.

Outcome and Prognosis

The mortality rates from SEA continue to decrease. Although mortality rates have been reported to be between 13 and 23 % [1, 2], lower rates of mortality are anticipated with prompt surgical decompression and tailored antibiotic therapy. Prior to the availability of antibiotics, mortality exceeded 50 %. Despite better imaging modalities and surgical techniques and equipment, a large percentage of patients suffer significant neurological deficits despite treatment chiefly due to diagnostic delay [45]. The strongest predictors of unfavorable outcome include neurological deficits at presentation, the duration of neurological deficits prior to surgery, and the severity of the neurological deficits. Deficits lasting >48 h have an unfavorable outcome [1, 2, 5, 11]. Other factors proposed as predictors of poor outcome include age, prior spinal surgery, low platelet count, elevated ESR >110, and cervical location [2, 46, 58•]. In recent series, about 75 % of patients with SEA who have been surgically decompressed have either recovered fully or have exhibited only a minimal weakness [1, 3, 63–66].

Conclusion

Early diagnosis of SEA prior to development of neurological deficits is imperative. Among patients presenting with only back pain, screening for other risk factors and obtaining inflammatory markers should be considered to identify candidates who merit MRI. In patients diagnosed with SEA, early surgical intervention and drainage of the abscess with concomitant antibiotic treatment appears to be the most effective treatment strategy, although select patients may be managed by antibiotic therapy and conservative measures alone if closely monitored for any clinical deterioration or signs of failure of medical management.

Compliance with Ethics Guidelines

Conflict of Interest Joseph Berger received grants from PML Consortium, Biogen Idec, and Novartis. Berger received personal fees from Amgen, Astra-Zeneca, Bristol Myers Squibb, Eisai, Janssen, Millennium, Parexel, Pfizer, Roche, Takeda, Genentech, Genzyme, Incyte, Inhibikase, Johnson and Johnson, Novartis, the American Academy of Neurology, and the Consortium of MS Centers. Prashanth Krishnamohan has no conflicts of interest.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by the author.

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