Intern Emerg Med (2017) 12:1179–1183
DOI 10.1007/s11739-017-1718-5

EM - REVIEW

The time-sensitive challenge of diagnosing spinal epidural abscess in the emergency department

Stephen Alerhand[1] · Sumintra Wood[1] · Brit Long[2] · Alex Koyfman[3]

Received: 2 March 2017 / Accepted: 29 July 2017 / Published online: 4 August 2017

Abstract Spinal epidural abscess (SEA) is a rare but devastating condition. Entry of infectious contents into the epidural space occurs via contiguous infected tissue, hematogenous spread, or iatrogenic inoculation. Traditionally, emergency providers are taught to assess for the "classic triad" of spinal pain, fever, and neurological deficits, but this constellation of findings is seen in only 10–15% of cases. Delays in diagnosis and treatment of this condition directly correspond to worse, and often debilitating, outcomes for these patients. This review will demonstrate the challenges of diagnosing SEA, describe key diagnostic pitfalls, and present a model and framework for its evaluation. The authors conducted a systematic review in PubMed and Google Scholar for articles describing the emergency medicine evaluation and management of spinal epidural abscess dating from 1996 to 2016. Of the initial 219 articles found, 18 articles were selected based on their relevancy to emergency medicine. Lower back pain is a common chief complaint, whereas SEA is a rare condition and may not be anticipated. The "classic triad" of SEA symptoms presents infrequently. Moreover, the early symptoms of back pain and fever are non-specific, and patients seek medical attention at varying stages of disease progression. Once the more conspicuous and wide-ranging neurological symptoms develop, they are often irreversible. In fact, final outcomes correlate with the severity and duration of symptoms before surgery. Furthermore, discovering these late neurological symptoms can be particularly difficult in bed-bound and chronically ill patients. MRI is the best diagnostic imaging tool for SEA. Early diagnosis is the major prognostic factor for favorable outcome of SEA, and yet, making this diagnosis in the emergency department (ED) has proved challenging. Shifting from a "classic triad" screening to a risk factor-based model of evaluation represents the current optimal strategy for diagnosing SEA. An algorithm incorporating the most recent data is provided.

Keywords Spinal epidural abscess · Spinal infection

✉ Stephen Alerhand
Stephen.Alerhand@gmail.com

Sumintra Wood
sumintron@gmail.com

Brit Long
brit.long@yahoo.com

Alex Koyfman
akoyfman8@gmail.com

[1] Department of Emergency Medicine, Icahn School of Medicine at Mount Sinai, 1 Gustave L. Levy Place, New York, NY 10029, USA

[2] Department of Emergency Medicine, San Antonio Military Medical Center, 3841 Roger Brooke Dr, Fort Sam Houston, San Antonio, TX 78234, USA

[3] Department of Emergency Medicine, The University of Texas Southwestern Medical Center, 5323 Harry Hines Boulevard, Dallas, TX 75390, USA

Introduction

Spinal epidural abscess (SEA) is a rare but potentially devastating diagnosis that can be difficult to identify in the emergency department (ED). Unfortunately, once the classic symptoms appear and a definitive diagnosis is made, the severe neurologic deficits already present (including motor paralysis and sensory weakness) may be irreversible [1].

Incidence, epidemiology, and risk factors

SEA was previously reported to occur in 0.2–1.2 patients per 10,000 hospital admissions in 1975 by Baker et al. [2]. It has lately risen to 2.5–3 patients per 10,000 admissions due to an increase in predisposing conditions and spinal instrumentation [3]. SEA develops via entry into the epidural space from contiguous infected tissue (vertebral body, psoas muscle), hematogenous spread (skin, soft tissue, urinary, and respiratory tract infections), or iatrogenic inoculation (epidural analgesia, paraspinal steroid injection, lumbar puncture, surgery, nerve block). No source for the abscess is identified in 30–40% of cases [4, 5]. The collection of pus or inflammatory granulation grows between the dura and vertebral column, usually in the thoracic and lumbosacral regions that are wider and contain more infection-prone fat tissue. Typically, these abscesses extend an average of four vertebrae [4]. Damage to the spinal cord can be caused not only by direct compression, but also from thrombosis and thrombophlebitis of nearby veins, interruption of arterial blood supply, and bacterial toxins and inflammatory mediators [6].

Risk factors for spinal epidural abscess are diverse and include: spinal instrumentation, contiguous bony or soft tissue infection, bacteremia secondary to distant infection, controlled or uncontrolled diabetes mellitus, immunocompromise (AIDS, immunosuppressive therapy, malignancy), trauma, history of IV drug abuse, dialysis, steroid use, and alcoholism [1, 7]. The overarching theme of these risk factors is that they raise the patient's risk for bacteremia, and this correlates with the pathophysiology of the disease as described above. Of note, the risk of SEA is very low with temporary invasive spinal punctures such as with lumbar puncture, but it increases significantly with indwelling peri- or epidural catheter duration of at least 2–4 days [8, 9]. Irrespective of these risk factors, the "classic triad" of presenting symptoms commonly associated with SEA consists of spinal pain, fever, and neurologic deficits [10], but by no means are these always present.

Radiographic diagnosis

Definitive diagnosis is best achieved using gadolinium-enhanced MRI imaging, which carries both a sensitivity and specificity above 90% [11, 12]. If MRI is not readily available, the next best imaging modality is a CT with IV contrast. CT myelogram is nearly as sensitive as MRI for detecting SEA, but the more invasive nature of the study and the potential risk of spreading infection to the subarachnoid space limits the use of this imaging modality [13].

Laboratory adjuncts

Laboratory studies can assist in the diagnosis of SEA, but are far from specific for this condition. An ESR >20 mm/h is found in at least 94% of cases [1], and elevation is present in all but one of 63 SEA patients in a study by Davis et al. [10]. However, though sensitive, neither ESR nor CRP are specific for SEA [10]. White blood cell count may be elevated or be normal [10]. Blood cultures should also be obtained in all cases of suspected SEA as they are positive in 60% of cases [13]. Cultures obtained from the abscesses themselves are positive in >90% of cases, with *Staphylococcus aureus* being the most frequently identified pathogen (63%) [13]. Note that lumbar puncture typically should not be performed in cases of suspected or confirmed SEA, as the procedure may serve to further seed the infection. Lumbar imaging with CT scan may also be considered beforehand if the patient has a known spinal deformity or history of spinal fusion. Cerebrospinal fluid does not reliably yield positive cultures either [14].

Management and prognosis

Management of SEA involves abscess drainage and eradication of the causative organism. After obtaining blood cultures plus any additional cultures from possible sources of infection, broad-spectrum empiric antibiotics should be administered. There are no randomized controlled trials evaluating the efficacy of different regimens for treating this condition, but Vancomycin plus Cefepime plus Metronidazole is a reasonable combination for targeting of staphylococci, streptococci, and gram-negative bacilli. Most cases of SEA require surgical decompression by laminectomy, hemilaminectomy, or interlaminar fenestration. CT-guided needle aspiration combined with antibiotics may be beneficial in patients with posterior SEA, no neurological deficit, high surgical risk, or who do not respond to antibiotics alone. Antibiotics alone are an option for patients who represent a high surgical risk, or if there has already been a prolonged duration of symptoms. Of high importance, neurological improvement is unlikely if pre-intervention paresis has exceeded 24–36 h [14]. Conversely, factors associated with improved neurologic outcome in one retrospective study include age <60 years, no comorbidities, and abscess size (<50% thecal sac compression) [15].

Methods

The authors independently conducted a systematic review in PubMed and Google Scholar for relevant articles concerning spinal epidural abscess dating from 1996 to 2016.

Terms included spinal epidural abscess, evaluation, management, spinal infection, back pain, diagnosis, imaging, antibiotics, antimicrobials, neurologic deficit, magnetic resonance imaging, fever, emergency department, and emergency medicine. Authors focused on studies investigating the ED evaluation and management.

Search criteria utilized English language for study inclusion, with primary focus on studies relevant to emergency medicine. Studies further than 20 years ago were excluded to focus on recent literature information, whereas the wide time period aimed to compensate for the extremely rare incidence of SEA by capturing sufficient data. Primary literature and reviews were included. Once the initial literature search was completed, titles and abstracts were screened by two authors for relevance to ED evaluation and management. Following this step, the full manuscripts of the pertinent studies were obtained and thoroughly evaluated. Studies were selected based on the following characteristics clinical or emergency medicine evaluation and management.

Results

Of the initial 219 articles found in PubMed and Google Scholar, 18 articles were selected based on their relevancy to emergency medicine.

SEA is difficult to diagnose

Although the definitive radiographic diagnosis and subsequent management of SEA is well illustrated, the initial diagnosis of SEA can be challenging, given the rarity of the disease and the relatively non-specific nature of initial symptoms. In a retrospective study of 63 patients with SEA, diagnostic delays (multiple ED visits before diagnosis, admission without diagnosis of SEA, >24 h to a definitive study) are present in 75% of SEA patients [10].

Reasons for missing the diagnosis

To begin with, the "classic triad" of SEA symptoms is infrequently present. It was found in only 10–13% of cases at the first physician contact in Davis et al.'s study [10]. Fever was measured in only 32% of cases at the first ED visit, and the neurological examination was documented as normal in 68%. In a review of 75 cases, Rigamonti et al. found the "classic triad" present in 37%, while 22% have no neurologic deficit with or without back pain [16]. Over a range of studies, diffuse spine pain and severe focal spinal tenderness are the most frequent early findings at 65 and 52%, respectively [10].

Moreover, patients with SEA present to the ED at variable stages of disease progression. For Davis et al., the mean duration of symptom onset before the first ED visit and at admission was 5 and 9 days, respectively, while the median number of ED visits before SEA diagnosis was 2 [10].

Additionally, the neurological deficits associated with SEA can be wide-ranging. Motor symptoms can range from focal motor signs along a specific dermatome to complete paraplegia. Sensory symptoms may include paresthesias, hyperesthesias, and a pressure-like sensation. It may also be difficult to pick up on "hidden" neurological symptoms in patients who are bed-bound or ill with other conditions.

Given that SEA itself is an extremely rare diagnosis, whereas low back pain is the 5th most common reason for physician visits, providers may often misattribute initial symptoms of SEA to musculoskeletal pain. Complicating the diagnostic work-up of concerning lower back pain, MRI is not always readily available within EDs. Laboratory markers have also proven to be of limited value for the diagnosis of SEA, although inflammatory markers may have promised.

Consequences of missing the diagnosis

SEA presents a significant diagnostic challenge, and carries very poor outcomes when missed. Cases with a delay in diagnosis have an increased risk of residual weakness versus those with no delay (45 vs. 13%) [10]. Duration and severity of neurological deficits at the time of diagnosis also correlate with patients' ultimate neurological function. Almost 50% of survivors emerge with residual neurologic deficits, including 15% with paresis or complete paralysis [1]. Thus, minimizing delay to diagnosis of SEA is crucial for improving patients' morbidity and mortality. Death from SEA usually occurs due to severe overwhelming sepsis, and is more likely to occur in patients with multiple comorbidities, with mortality rates between 2 and 20% [14].

Discussion

Given the previously noted pitfalls and lack of high-quality evidence stemming from low incidence, the following represents a current optimal strategy for evaluating patients considered for SEA.

Recommendations for clinical evaluation

A more reliable strategy to assess for SEA is needed in order to minimize diagnostic delay and thereby decrease morbidity and mortality (Table 1).

Table 1 Clinical recommendations to assess for SEA in the ED

Screening	Diagnosis and management
Risk factor assessment over "classic triad"	Emphasis on early diagnosis
Consider ESR as screening tool	Urgent MRI for high-risk patients
	Early surgery unless contraindicated

SEA spinal epidural abscess, *ESR* erythrocyte sedimentation rate

Table 2 Risk factors for SEA

Sources for infection	Immunosuppression
Recent spinal instrumentation	Diabetes mellitus
IV drug use	AIDS
Hemodialysis	Malignancy
Contiguous bony or soft tissue infection	Immunosuppressive therapy
Bacteremia from distant infection	Steroid use

SEA spinal epidural abscess

Early diagnosis is the major prognostic factor for a favorable outcome. No matter what is the provider's experience or education level, SEA must at least be on the differential for back pain, especially in patients who have received recent spinal instrumentation indwelling at least 2–4 days. Furthermore, based on the "classic triad's" relative lack of sensitivity, a risk factor assessment should instead be used to guide the emergency physician's clinical judgment (Table 2). A complete neurologic examination must also be performed including sensory and motor function, reflexes, and gait. Point-of-care ultrasound may also be used to measure post-void residual urine. Healthy adults usually feel the first need to void at a bladder volume of 150 mL and the urge to void at 300 mL [17]. Physicians must be especially vigilant with bed-bound patients and those with other debilitating comorbidities.

Given the high sensitivity of ESR for SEA (elevated in at least 94% of cases [1] and up to 100% in one study [18]), an ESR level <20 mm/h might be useful as an early screening tool in select patients with spinal pain but without neurological deficits. Radiologic imaging including consideration for emergent MRI should be obtained for high-risk patients with neurological deficit and focal back pain, deficit and unexplained fever, deficit and elevated ESR, severe focal back pain and fever, severe focal back pain with markedly elevated ESR, or unexplained severe focal back pain [18]. Lastly, the emergency physician caring for a patient with an MRI-diagnosed SEA should advocate for their patient's early surgical intervention unless otherwise contraindicated.

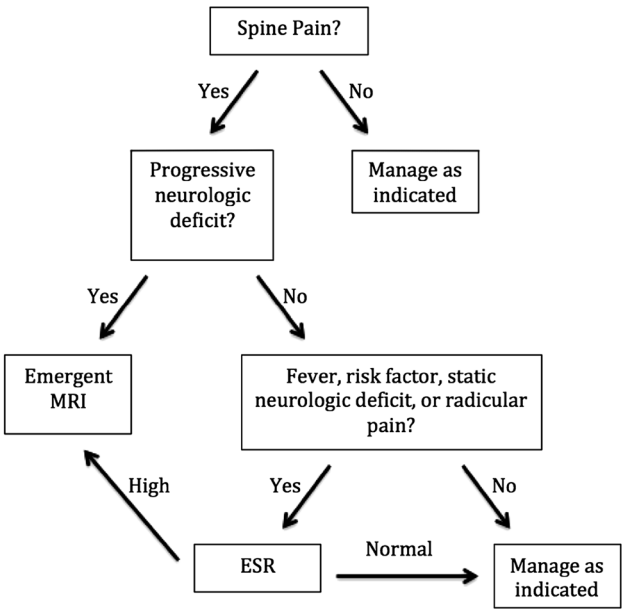

Fig. 1 Decision guideline for SEA using risk factor assessment followed by ESR testing prior to definitive imaging. *SEA* spinal epidural abscess, *ESR* erythrocyte sedimentation rate

Consideration of a proposed diagnostic algorithm

When compared to a control group of 55 patients with SEA, an evaluation and treatment paradigm developed by Davis et al. shows improvement in making the diagnosis [18] (Fig. 1). The rate of diagnostic delay decreases (from 83.6 to 9.7%), and ESR appears to be a highly sensitive (100%) and moderately specific (67%) intermediate screening tool in those patients with spinal pain and an SEA risk factor. The importance of this improvement in timely diagnosis is highlighted by the decreased incidence of motor deficits at time of diagnosis (from 81.8 to 19.4%).

The decision guidelines in the Davis study do not mandate immediate MRI in all patients with a SEA risk factor and an elevated ESR and CRP. However, information on MRI utilization is not delineated in the study, as acknowledged by the authors. It can be assumed that with a higher usage rate based on the algorithm's inclusion criteria, the amount of negative MRI results would

correspondingly increase. Many EDs do not have easy and rapid access to MRI imaging, so when incorporating cost and resources, it becomes clear that increased MRI usage for suspected SEA as per the algorithm carries certain logistical limitations.

Overall, the combination of the provider's clinical judgment and the decision guideline (including its selective use of ESR for further stratification) may provide a framework with which to evaluate patients with symptoms concerning for SEA. As mentioned earlier, the sensitivity and specificity of MRI are above 90%, thus those facilities without access to this imaging modality may consider transfer to another facility that has access to that imaging modality. Patients in whom SEA is clinically probable, and yet have a negative MRI, ought to be observed very closely, with antibiotics administered in the meantime concurrent with a neurosurgical consultation.

Conclusions

The most important prognosticator for favorable outcomes in patients with SEA is making the diagnosis as early as possible. Unfortunately, achieving a rapid diagnosis in the ED has proved challenging, and the delay in making the diagnosis is often accompanied by devastating neurologic deficits. Rather than evaluating for the "classic triad" of symptoms that is not often present, implementing a risk factor screening for SEA represents the current optimal strategy for early diagnosis.

Acknowledgements This manuscript did not utilize any grants, and it has not been presented in abstract form.

Compliance with ethical standards

Conflicts of interest All authors declare that they have no conflicts of interest.

Statement of human and animal rights This article does not contain any studies with human participants or animals performed by any of the authors.

Informed consent Informed consent is not required.

References

1. Reihsaus E, Waldbaur H, Seeling W (2000) Spinal epidural abscess: a meta-analysis of 915 patients. Neurosurg Rev 23(4):175–204 **(discussion 205)**
2. Baker AS et al (1975) Spinal epidural abscess. N Engl J Med 293(10):463–468
3. Sampath P, Rigamonti D (1999) Spinal epidural abscess: a review of epidemiology, diagnosis, and treatment. J Spinal Disord 12(2):89–93
4. Darouiche RO et al (1992) Bacterial spinal epidural abscess. Review of 43 cases and literature survey. Med (Baltim) 71(6):369–385
5. Danner RL, Hartman BJ (1987) Update on spinal epidural abscess: 35 cases and review of the literature. Rev Infect Dis 9(2):265–274
6. Scheld WM, Whitley RJ, Durack DT (1997) Infections of the central nervous system, 2nd edn. Lippincott-Raven, Philadelphia, p 1064, xix
7. Reynolds F (2008) Neurological infections after neuraxial anesthesia. Anesthesiol Clin 26(1):23, v–52, v
8. Christie IW, McCabe S (2007) Major complications of epidural analgesia after surgery: results of a six-year survey. Anaesthesia 62(4):335–341
9. Cameron CM et al (2007) A review of neuraxial epidural morbidity: experience of more than 8000 cases at a single teaching hospital. Anesthesiology 106(5):997–1002
10. Davis DP et al (2004) The clinical presentation and impact of diagnostic delays on emergency department patients with spinal epidural abscess. J Emerg Med 26(3):285–291
11. Angtuaco EJ et al (1987) MR imaging of spinal epidural sepsis. AJR Am J Roentgenol 149(6):1249–1253
12. Wong D, Raymond NJ (1073) Spinal epidural abscess. N Z Med J 1998(111):345–347
13. Curry WT Jr et al (2005) Spinal epidural abscess: clinical presentation, management, and outcome. Surg Neurol 63(4):364–371 **(discussion 371)**
14. Sendi P, Bregenzer T, Zimmerli W (2008) Spinal epidural abscess in clinical practice. QJM 101(1):1–12
15. Khanna RK et al (1996) Spinal epidural abscess: evaluation of factors influencing outcome. Neurosurgery 39(5):958–964
16. Rigamonti D et al (1999) Spinal epidural abscess: contemporary trends in etiology, evaluation, and management. Surg Neurol 52(2):189–196 **(discussion 197)**
17. Buchko BL, Robinson LE (2012) An evidence-based approach to decrease early post-operative urinary retention following urogynecologic surgery. Urol Nurs 32(5):260–264 **(273)**
18. Davis DP et al (2011) Prospective evaluation of a clinical decision guideline to diagnose spinal epidural abscess in patients who present to the emergency department with spine pain. J Neurosurg Spine 14(6):765–770