Q J Med 2008; **101**:1–12
doi:10.1093/qjmed/hcm100

Review

QJM

Spinal epidural abscess in clinical practice

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Summary

Spinal epidural abscess (SEA) is a rare but severe infection requiring prompt recognition. The major prognostic factor for a favourable outcome is early diagnosis, leading to appropriate treatment. In clinical practice, a diagnosis of SEA is often not considered, particularly in the early stages of the disease when neurological symptoms are not apparent. Knowledge of persons at risk, clinical features and the required diagnostic procedures may decrease the number of initially misdiagnosed cases. Clinical signs, duration of symptoms and the rate of neurological deterioration show a high inter-individual variability, and the classic triad (spinal pain, fever and neurological deficit) is often not found, especially not at first presentation to a physician. However, most patients complain of severe localized back pain. Inflammatory parameters in the blood are generally elevated, but not specific. Gadolinium-enhanced magnetic resonance imaging is the most sensitive, specific and accurate imaging method. Although neurosurgical decompression is still the treatment of choice in the majority of cases, less invasive procedures (e.g. computed tomography-guided needle aspiration) or antimicrobial treatment alone can be applied in selected cases. The choice of the most appropriate therapy should be discussed immediately after a confirmed diagnosis in consultation with infectious disease, radiology and spinal surgery specialists. The outcome of SEA is largely influenced by the severity and duration of neurological deficits prior to surgery, stressing the importance of early recognition.

Introduction

Spinal epidural abscess (SEA) represents a diagnostic challenge in clinical practice. Prior to the appearance of neurological deficits, its signs and symptoms are applicable to a broad range of possible diagnoses. The combination of low incidence and non-specific symptoms such as back pain or localized spinal tenderness can make early recognition difficult. Unrecognized SEA may not only progress to potentially irreversible pareses, but also to life-threatening sepsis. A high degree of awareness can lead to rapid detection of SEA and thus a better prognosis. Clinicians assessing a patient at an early stage need to initiate the correct diagnostic work-up promptly, and so internists, primary care physicians and emergency medicine physicians are the target readers of this review.

Epidemiology

In 1975, Baker *et al.*[1] reported an incidence of SEA ranging from 0.2 to 1.2 per 10 000 hospital admissions per year. Since then, several studies have shown that the incidence is rising. The current annual incidence is estimated to be 2.5–3 per 10 000 hospital admissions.[2–10] This trend can be

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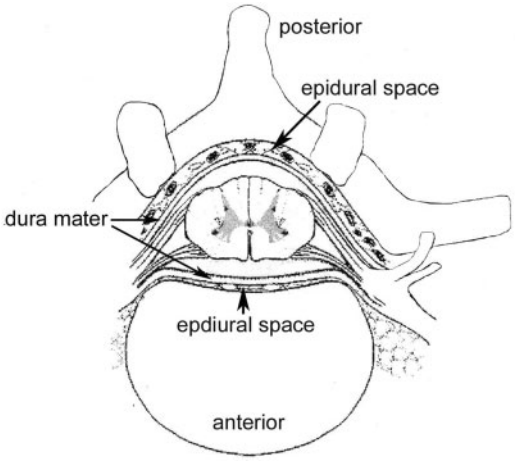

Figure 1. Cross-section through a vertebra. Epidural space.

partly explained by the growing number of patients with predisposing conditions or risk factors, such as diabetes mellitus, higher age or intravenous drug use. Instrumentation of the vertebral canal by anaesthetic interventions is also a risk factor contributing to the incidence rate of SEA. Part of the increase may also be artefactual, and due to the improved sensitivity of the now broadly available neuroradiological imaging techniques. It is conceivable that previously, in some patients, epidural abscesses were not recognized, and were diagnosed and treated as spondylitis.

SEA occurs in all age groups.[11] However, a greater prevalence between the fifth and seventh decade of life[3–8,12] and a male predominance[4,6–8,11] have been described in many studies. This might be due to the fact that the reported predisposing conditions and risk factors are more prevalent in older people.

Key points for clinical practice

The annual incidence of SEA is estimated to be 2–3 per 10 000 hospital admissions. Prevalence is greatest between the fifth and seventh decades of life.

Anatomy

Spinal epidural infection is characterized by a collection of pus or inflammatory granulation tissue between the dura mater and the overlying vertebral column. The spinal epidural space (Figure 1) is a continuous vertical sleeve largely filled with fat, arteries and venous complexes. In the lumbar region, however, the dura approaches the periosteum, giving the appearance of a segmented sleeve. There is a true epidural space posterior and lateral to the spinal cord. In contrast, the anterior area is almost virtual, since the dura, the posterior longitudinal ligament and the periosteum of the vertebral body are in close contact with each other.

The posterior area is small in the cervical region, but larger in the midthoracic (T4–T8) and lumbosacral (L3–S2) part of the spine. It is plausible that the location and extent of SEA are associated with anatomical structures. As the abscess increases in size, it extends along the dural sheath, and therefore usually involves several segments.[3–7,13,14] SEA is typically located in the 'true' posterior space. Abscesses located anterior of the spinal cord are frequently but not exclusively associated with vertebral osteomyelitis.[3,5,6] In most studies, SEA is predominantly located in the thoracic and lumbosacral region.[3,4,6,7,11,14,15] This parallels the most frequent localization of vertebral osteomyelitis, and the preferred puncture site of instrumentation of the vertebral canal.[16,17]

Key points for clinical practice

SEA usually involves several segments. SEA is predominantly located in the posterior thoracic and lumbosacral spine. Abscesses located in the anterior epidural space are frequently associated with vertebral osteomyelitis.

Pathogenesis

Bacteria gain access to the epidural space by three mechanisms: (i) per continuitatem from a neighbouring infected structure; (ii) through haematogenous dissemination from a remote focal infection; or (iii) through iatrogenic inoculation. However, in 30–40% of the cases, no source can be identified, indicating silent bacteraemia seeding in the epidural space.[3,4,18]

Direct extension usually originates from vertebral osteomyelitis or psoas muscle abscess. This mechanism is estimated to be responsible for 10–30% of cases.[3,11,18] Thus, it is important to evaluate and monitor the extent of the primary infection.

In 50% of cases, microorganism reach the epidural space through haematogenous seeding. Skin, soft-tissue, urinary and respiratory tract infections are frequent primary sources (Table 1). Importantly, as a result of haematogenous dissemination, SEA may occur discontinuously at several levels of the spine. This should be taken into consideration when spinal tenderness is assessed and imaging studies planned.

Iatrogenic causes of SEA include all kinds of invasive procedures, such as surgery, lumbar puncture, peridural anaesthesia, epidural analgesia

Table 1 Primary sources of infection in spinal epidural abscess

Source of infection	Median (%)	Range (%)
Skin and soft tissue	18	7–45
Urinary tract	10	2–36
Previous sepsis of unknown origin	8	5–11
Respiratory tract	5	3–16
Abdomen	4	2–11
Endocarditis	3	1–8
Infected vascular access	2	1–8
Dental abscess	2	1–11
Ear, nose and throat	2	<1–11

Based on references 1, 3–7, 11, 18–22.

Table 2 Predisposing conditions in spinal epidural abscess

Predisposing condition	Median (%)	Range (%)
Diabetes mellitus	21	15–46
Abnormality of the vertebral column	17	6–70
Trauma of the spine	15	5–33
Intravenous drug use	15	4–37
Immunosuppressive therapy	12	7–16
Cancer	7	2–15
HIV/AIDS	6	2–9
Alcoholism	5	4–18
Chronic renal failure	4	2–13

Based on references 1, 3–7, 11, 18–22.

and nerve blocks, and are estimated to be responsible for ~15% of all cases.[1,3–7,11,18–23] In general, these infections are acquired either during the invasive procedure itself, or through ascending microorganism from the skin flora, when a device is left in place.[24–26] On such catheters, there is a biofilm formation similar to that found on intravascular catheters. Another possible iatrogenic cause of SEA that clinicians should be aware of, is the paraspinal injection of analgesics and steroids (e.g. for local pain therapy).[13,27–29]

Spinal cord injury leading to neurological impairment is partially caused by direct mechanical compression by the inflammatory mass. Accordingly, there is notable neurological improvement after surgical decompression.[30] Studies focussing on the indirect injury caused by vascular occlusion and ischaemia have shown diverging results.[31,32] Mechanical compression and vascular occlusion may occur at different phases of the disease and cause additive adverse effects. However, the detailed pathogenesis of spinal cord injury remains poorly characterized.

Key points for clinical practice

SEA can occur simultaneously on several segments of the spine. In case of severe spinal tenderness occurring during or after any focal infection or sepsis, SEA must be considered as a diagnosis. In the case of vertebral osteomyelitis or psoas muscle abscess, SEA must be actively looked for. Previous invasive spinal procedures, including paravertebral injections of analgesics and steroids for local pain therapy, represent a possible source of infection.

Predisposing conditions and risk factors

A large proportion of SEA patients have at least one predisposing factor (Table 2). Most of these, including diabetes mellitus, intravenous drug use, immunosuppressive therapy, cancer, HIV/AIDS and renal failure are predisposing conditions for any type of severe infection. Spinal abnormalities, such as degenerative joint disease or scoliosis, have been advocated to represent a *locus minoris resistentiae*. A history of previous spinal trauma is often evident in SEA. Haematoma and disruption of anatomic barriers favour the development of SEA.[11]

Alcoholism is found in a relatively high proportion of patients with SEA. Alcohol intoxication predisposes to injury, including spinal trauma, and decreases pain sensitivity, resulting in pressure sores or muscle damage. Moreover, there is a high risk for missing the diagnosis of SEA in this population, because symptoms might be misinterpreted as typical sequelae of alcoholism, such as pancreatitis, peripheral neuritis, and vitamin B12 deficiency.[20]

The risk of SEA in association with invasive procedures has been estimated for some invasive anaesthetic interventions and ranges from 1:1000 to 1:100 000, depending on the study population, and the location and duration of catheterization.[26,33–40] In the case of temporary puncture, the risk of an epidural abscess is very low. Two recently published studies,[23,41] each analysing the outcome of >8000 epidural catheters inserted for postoperative analgesia, calculated an SEA incidence of approximately 1:1350. However, if a peri- or epidural catheter is left in place for several days (e.g. for more than 2–4 days), the risk of developing both catheter site infection and epidural abscess increases

significantly.[23,41,42] The true incidence of SEA after paravertebral injection for pain therapy, in particular when performed repeatedly, is difficult to establish, because the frequency of this debated practice and its complications may be underreported. Thus, it might be possible that these interventions outnumber other iatrogenic causes of SEA.[29,43] However, preceding anaesthetic practices must be actively sought when evaluating the patient's history.

Key points for clinical practice

Awareness of co-morbidities can speed up the identification of patients prone to infections. Spinal abnormalities and a history of spine trauma are predisposing conditions for SEA. The risk of acquiring SEA during anaesthetic practices is low, but increases with time when peri- or epidural catheters are left in place. Preceding anaesthetic practices must be actively sought when evaluating a patient's history.

Clinical features

Many clinical features are non-specific, even in an emerging case of SEA, particularly if the circumstances are not considered. However, if a thorough evaluation of the patient's history reveals predisposing or risk factors for SEA, the constellation of several symptoms and signs should raise the suspicion of this diagnosis.

Classical symptoms of SEA include spinal pain, fever and neurological deficits. However, according to two recent studies, this triad is only present in 10–15% of the cases at first physician contact.[44,45] Common reported symptoms and signs at the time of diagnosis are listed in Table 3. Back pain and severe, circumscribed tenderness are by far the most frequent early findings. Thus, patients are at risk of receiving inappropriate treatment such as analgesics for back pain due to degenerative spinal disease. However, many patients describe the pain as 'the worst they ever had',[18] and are able to distinguish the pain character from chronic back pain. When asked about the ache type, some patients report sharp or lancinating pain, like 'being stabbed in the back'.[8,18] Nevertheless, since the differential diagnosis of back pain is large, and the pain type does not reliably indicate SEA, diagnosis of SEA is still likely to be missed, particularly at early stages.[1,4] Therefore, considering a diagnosis of SEA in the differential diagnosis is crucial for early detection and outcome. This also holds true for patients with dominating signs of sepsis, in whom neurological symptoms may not be noticed, although the combination of fever, meningism and/or neurological deficits might occur more frequently at late stages of the disease. Importantly, a thorough neurological examination, including the evaluation of reflexes, sensory and motor functions, anal sphincter tonus and the ability to completely void the bladder is mandatory in patients presenting with severe localized back pain. Special attention should be paid to the extremities and dermatomes corresponding to the affected spine level. Grading the muscle activity (i.e. British Medical Council M-scale, M5 to M0) can also be helpful to follow the motor function during the course of the disease.

In 1948, Heusner summarized the clinical features and progression of SEA in four stages (Table 4).[46,47] This staging system is still a valuable tool, because it potentially points to the diagnosis of SEA before the appearance of irreversible neurological damage. However, additional information is needed for clinical practice. In stage II, signs of nerve root compression may present as typical radicular pain, but have also been described as

Table 3 Symptoms and signs at diagnosis of spinal epidural abscess

Signs/symptom	Median (%)	Range (%)
Back pain	75	72–100
Tenderness	58	17–98
Motor weakness	40	26–87
Radicular pain	38	19–57
Sensory abnormalities	36	13–45
Fever	32	13–95
Bladder and bowel dysfunction	27	9–37
Paralysis	27	5–39
Neck stiffness	16	2–24
Confusion	14	7–28
Headache	14	3–24
Nausea/vomiting	8	1–8

Based on references 1, 4–6, 8, 11, 14, 18, 19, 55, 70, 71.

Table 4 Stages according to clinical progression of spinal epidural abscesses

Stage	Clinical signs
I	Back pain, fever, tenderness
II	Radicular pain, nuchal rigidity/neck stiffness, reflex changes
III	Sensory abnormalities, motor weakness, bowel and bladder dysfunction
IV	Paralysis

Based on references 46, 47.

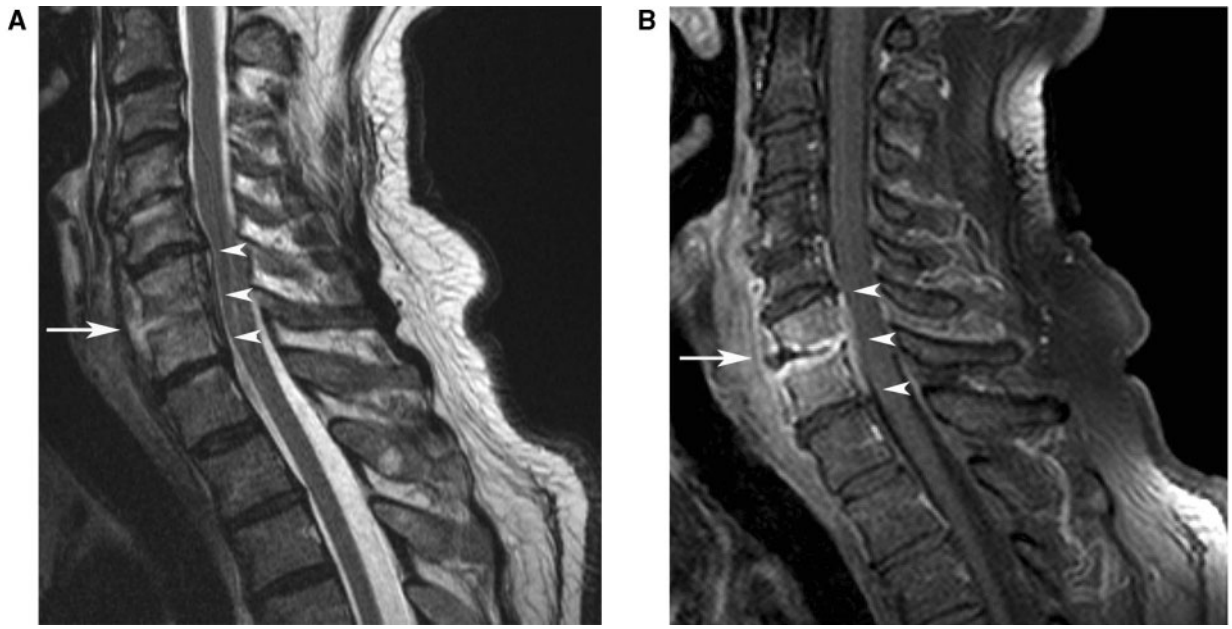

Figure 2. MRI. T2-weighted image (**A**) and T1-weighted fat-saturated image after intravenous gadolinium (**B**), showing spondylodiscitis (arrow) complicated by an SEA at the C6–7 level (arrowheads).

'electric shocks' or 'shooting'.[1] Depending on the spine level involved, the pain can radiate to the abdomen, chest wall or neck, and mimic other diseases such as pancreatitis or heart disease.[20,48] Sensation alterations such as numbness may already indicate stage III and should alert the physician to the severity of the disease. Subtle signs of motor weaknesses, bowel and bladder dysfunction may not be realized or reported by the patient, and must be actively looked for, as discussed above. Once complete loss of muscle activity (paralysis) develops, stage IV according to Heusner, it quickly becomes irreversible, stressing the seriousness of a delayed diagnosis.

The duration of symptoms and the rate of neurological deterioration are highly variable.[3,4,30] The time between symptom onset and the patient consulting a physician or appearing in the emergency department can range from a few days to more than a month.[19] Furthermore, neurological deterioration can progress to complete loss of muscle activity within a few hours, or alternatively to only slight impairments within several days.[1,4,6,49] This unpredictable course underlines the difficulty in diagnosing SEA, and the importance of repeating a thorough neurological examination frequently.

In contrast to other infectious diseases such as osteomyelitis, a subclassification into acute and chronic SEA is not clinically useful, because it shows poor correlation to specific aetiological agents, and does not significantly influence the choice of treatment or the outcome.[4,6,50,51]

Key points for clinical practice

SEA shows a large variability in clinical signs, duration of symptoms and the velocity of progression of neurological deficits. At an early stage of disease, the so-called classical triad (spinal pain, fever and neurological deficits) is not common. Severe localized back pain is the most frequent reported symptom and should induce more specific diagnostic tests. In suspected or identified SEA cases, frequent neurological examination is required.

Illustrative case report 1

A 62-year-old man was evaluated for the new onset of severe backache and fever. His medical history included psoriasis. Physical examination revealed no evidence of neurological deficits. Analyses of inflammatory parameters in the blood showed 8.2×10^9/l white blood cells (WBC, normal $4–10 \times 10^9$ cells/l) and 24 mg/l C-reactive protein (CRP, normal <5 mg/l). The values were interpreted as a response to a viral infection. Analgesic treatment included intramuscular application of diclofenac. However, the symptoms persisted. Two days later, WBC had increased to 12.0×10^9 cells/l and CRP to 300 mg/l. *Staphylococcus aureus* sepsis was documented and intravenous flucloxacillin started. Because of the severe cervical pain, magnetic resonance imaging (MRI) of the cervical spine was performed, and revealed spondylodiscitis complicated by an SEA at the C6-7 level (Figure 2, A and B).

Comment

Patients suffering from a chronic dermatological disease such as psoriasis have an increased risk for *Staphylococcus aureus* carriage, similar to patients with insulin-dependent diabetes.[52,53] This patient complained of severe back pain and fever without any localizing neurological symptoms. Fever and new-onset severe back pain combined with a risk factor for *Staphylococcus aureus* sepsis[54] motivated the specific diagnostic test (MRI) which revealed SEA prior to the appearance of neurological deficits.

Diagnosis

Laboratory studies

Inflammatory markers such as WBC count, CRP and erythrocyte sedimentation rate (ESR), are generally elevated. Leukocytosis is found in 60–80%, and an ESR >20 mm/h in up to 95% of cases.[4–7,14,18–21,55] These laboratory parameters might be supportive, but they are not specific for SEA. In Europe, measuring CRP has replaced ESR as a test to indicate (acute) inflammation. In the study of Soehle and Wallenfang,[5] the mean CRP of 25 patients with SEA was 150 ± 9 mg/l (normal <5 mg/l).

Imaging studies

MRI with gadolinium (Gd-MRI) has a specificity and sensitivity above 90% to detect SEA, and being superior to other imaging modalities, is therefore the diagnostic method of choice.[56–58] MRI is also the most sensitive and specific test for the diagnosis of infection in patients with low back pain, enabling detection of vertebral osteomyelitis.[59] It identifies signal abnormalities indicating spinal cord ischaemia and acute transverse myelopathy. In T1-weighted images, SEA and the spinal cord have the same intensity. SEA suppresses the signal from the cerebrospinal fluid (CSF) by gadolinium enhancement, and can thereby be anatomically localized.[60] The use of fat-saturated images allows the detection of additional bone marrow oedema and soft-tissue inflammation, and therefore the extent of the infection. In T2-weighted images, SEA often shows a signal increase, but there are variations in this pattern (Figure 2A), leading to possible difficulties when interpreting image findings.[61,62] Thus, it is helpful to compare T2- with T1-weighted images on which the infected area is enhanced by the contrast medium (Figure 2B).

MRI findings may also correlate with outcome. In a study including 18 patients, central stenosis of $\geqslant 50\%$ and an abscess length of >3 cm were significantly associated with a worse outcome.[63]

CT with intravenous contrast media has been proposed as an alternative diagnostic tool, although its sensitivity, specificity and accuracy are lower than those of MRI.[56–59] Myelography followed by CT is as sensitive as MRI, but has the following limitations. Compared to MRI, it is an 'invasive' intervention and exposes the patient to ionizing radiation.[6,8,55] Also, it typically shows a block to contrast medium flow at the level of the SEA, but cannot identify the cause of the thecal sac compression. Other lesions revealing similar radiological findings to those of SEA include epidural lipomatosis or cancer metastasis.[64,65] Furthermore, a complete block to contrast-liquid flow reveals only the upper or lower level, but not the full extent of the abscess. Finally, this procedure carries the risk of additionally contaminating the subarachnoidal space, with subsequent meningitis. Based on these arguments, myelography is generally no longer recommended. However, in very selected cases (e.g. patients who cannot undergo MRI examinations, further elucidation of inconclusive MRI or CT findings), myelography may represent an additional alternative.[66]

Other imaging methods (plain radiograph or nuclear scintigraphy) are not diagnostic for SEA, but may reveal indirect signs of concomitant spinal infections.

Repeating imaging studies, after initial negative findings

In a case of unremarkable or inconclusive MRI findings, but high clinical and laboratory suspicion for SEA, repeated imaging studies are required. However, the optimal method of, and interval until, the second investigation are both unknown. The few published data appear to favour CT-myelography,[6,8,18,67] which is meaningful if a definite diagnosis is required within a short period of time after the initial imaging study. However, in the absence of neurological deficits and urgency for surgical intervention, repetition of Gd-MRI might be preferable. The interval between the first and second image study has varied greatly in the cases we have investigated (range 0–16 days), and was mainly based on the clinical course.[67,68] What is important, however, is that imaging is repeated rapidly in a case of neurological deterioration or insufficient clinical and laboratory response to antimicrobial treatment.

Table 5 Infective agents in 508 patients with spinal epidural abscesses

Pathogen	%
Staphylococcus aureus	60
Gram-negative rods	10
Escherichia coli	3
Pseudomonas aeruginosa	2.5
Klebsiella spp.	1
Streptococcus spp.	9
Viridans group streptococci	2
Streptococcus agalactiae	1.5
Streptococcus pneumoniae	1.5
Enterococcus spp.	1
Coagulase-negative staphylococci	4.5
Anaerobes	2
Mycobacterium spp.	<1

Based on references 1, 3–8, 14, 18, 19, 22, 45, 55, 79, 88.

Microbiological studies

It is important to cultivate the causative microorganism, from blood and/or from the abscess. Since a large proportion of SEAs are caused by haematogenous dissemination, additional sampling from potential sources of infection may be beneficial. This is supported by the fact that in 10–15% of cases Gram-negative rods are isolated, and these are mostly associated with urinary tract infections.

CT-guided needle aspiration of the abscess should be performed early, because blood cultures remain negative in 40% of cases.[44,45] Specimens can also be obtained from surgical drainage for decompression. Previous antimicrobial therapy decreases the sensitivity of culture results. However, although adequate medical treatment requires identification of the causative pathogen, antibiotics should not be withheld simply to improve the sensitivity of the abscess culture, and the decision whether or not to administer antibiotics prior to an invasive intervention must be based on the patient's clinical condition (see Treatment below).

A large variety of pathogens have been found as causative agents for SEA, including mycobacteria, fungi and parasites,[11] but *S. aureus* is by far the most frequent. Table 5 summarizes the microbiology from 508 patients published in the literature.

Cerebrospinal fluid

Lumbar puncture plays a less important role in diagnosing SEA, and should not be performed routinely. Neither Gram staining (generally negative), nor cultures of CSF (growth in 6–28%) reveal results with acceptable sensitivity.[3,4,18,68] The cell count is generally elevated, but varies widely (20 up to several thousand cells/mm^3).[1,4,6,7,18–20,68,69] Chemical analysis almost uniformly shows a protein level of >0.45 g/l and a lactate value >2 μmol/l.[18,68] But although these findings are typical, they are not specific for SEA. However, in selected cases with a high clinical suspicion of SEA and unremarkable MRI, signs of parameningeal inflammation in the CSF may indicate a false-negative imaging study. In addition, the sensitivity and specificity of microbiological and chemical CSF analyses may be higher in the presence of encephalitis (4–35% of SEA cases).[1,4–6,8,11,14,18,19,55,70,71] Nevertheless, the potential risk of adverse events after lumbar puncture is undeniable, and awareness of them is clinically important. As the needle passes through the abscess, pathogens might migrate to the meninges or subdural space, resulting in expansion of the infection. Thus, the location and extent of the SEA (or suspected SEA) should be carefully evaluated on imaging studies for correct needle placement. Where there is an obstructive hydrocephalus or complete subarachnoid block, removal of CSF contains the risk of downward spinal coning.[72] Therefore, CT or MRI of the brain is mandatory prior to performing lumbar puncture. Taking all these considerations together, CSF should only be harvested in selected cases, or when myelography is indicated.

Key points for clinical practice

CRP is almost always elevated, but is not specific. Once SEA is considered, MRI is the imaging method of choice. In addition to blood cultures, which remain negative in 40% of the cases, CT-guided needle aspiration of the abscess should be attempted. Sampling from possible sources of infection should be included in microbiological studies. CSF should only be sampled in selected cases. In the CSF, pleocytosis and a high level of protein and lactate are typical, but not specific.

Illustrative case report 2

A 54-year-old insulin-dependent diabetic man was evaluated for back pain of 1 week duration. He was treated with oral analgesics without pain relief for 2 days. A CT scan of the thoracic spine revealed neither musculoskeletal pathologies, nor a focus of infection. Meanwhile, the patient suffered from fever, recurrent chills and paresis of the right arm. Analyses of inflammatory parameters in the blood showed WBC of 18.2×10^9/l (normal $4–10 \times 10^9$ cells/l) and a CRP of 288 mg/l (normal <5 mg/l). *S. aureus* grew in blood cultures, and parenteral

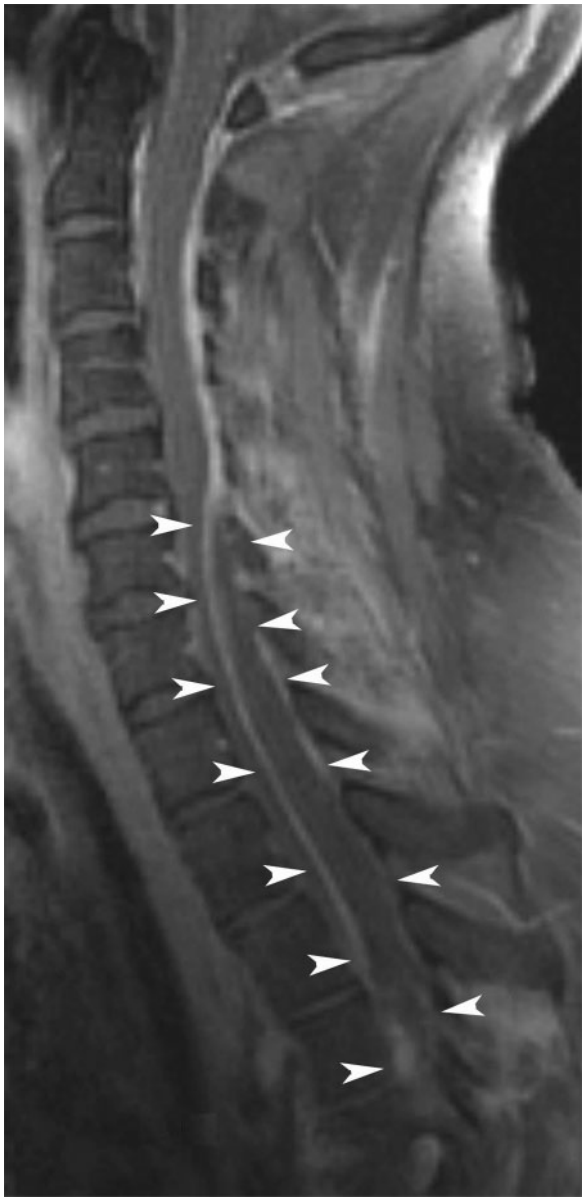

Figure 3. MRI. T1-weighted fat-saturated images after intravenous gadolinium, revealing a posterior SEA, from the vertebral level C5 to T5 (arrowheads).

antibiotics were administered. Two days after the CT scan, Gd-MRI revealed a SEA delineating from the vertebral level C5 to T5 (Figure 3).

Comment

In this febrile, diabetic patient with back pain, the diagnosis of SEA was initially not suspected. During the course of the disease, he presented with the classical triad, including back pain, fever and neurological deficits. The CT scan was done to search for musculoskeletal pathologies. It showed neither direct nor indirect signs of SEA. In view of the large extent of the abscess, it is likely that Gd-MRI would have revealed the diagnosis at first presentation. This case illustrates that Gd-MRI has a higher sensitivity than a normal CT scan and should therefore be the method of choice in SEA.

Treatment

The management of SEA should always be multidisciplinary and involve spinal surgeons, radiologists and infectious disease specialists. Drainage of the abscess and eradication of the microorganism are the basic principles of therapy. Treatment recommendations in SEA are largely based on retrospective studies, case series and expert opinions.

Empirical antimicrobial treatment

Antibiotics should be administered after cultures from blood and other possible sources of infection have been obtained. To increase the sensitivity of microbiological results, medical treatment can be slightly delayed and given immediately after the invasive procedure. In these cases, the absence of severe signs of infection and neurological deficits as well as prompt intervention are mandatory. Intravenous therapy must include anti-staphylococcal activity, as well as antimicrobial activity against streptococci and Gram-negative bacilli (e.g. amoxicillin/clavulanic acid, 2.2 g intravenously three times daily; or cefuroxime 1.5 g intravenously four times daily). Regional epidemiological data about methicillin-resistant *S. aureus* (MRSA) are decisive for the empiric use of glycopeptides. Previous history and clinical findings should influence the choice of agents. *Pseudomonas* spp. are associated with intravenous drug use. Coagulase-negative staphylococci are typically cultured in the presence of implanted devices, including epidural catheters. Once the causative pathogen is identified, antibiotic treatment should be streamlined accordingly.

Depending on the size of the abscess and the severity and duration of neurological deficits, the following three treatment options should be discussed interdisciplinarily.

Surgical decompression, drainage and antimicrobial therapy

Surgical therapy is the treatment of choice in the overwhelming majority of cases. Rapid surgical intervention is not only needed to minimize neurological damage, but also for controlling sepsis. The evaluation of the indication for decompressive surgical intervention should always urgently be

considered, since neurological improvement is unlikely if the duration of paresis exceeds 24–36 h. Based on the location and extent of the SEA, as well as intra-operative findings (e.g. use of intra-operative ultrasonography), the surgical approaches include laminectomy, hemilaminectomy or interlaminar fenestration.[3,8,9,12,18,29,70]

CT-guided needle aspiration combined with antimicrobial therapy

This diagnostic procedure might also be therapeutic by reducing the size of the inflammatory mass. To date, this method has only been reported in a few cases, and indication for this treatment is not clearly defined. Selected patients with a posterior SEA, no neurological deficit or high surgical risk, and who do not respond satisfactorily to antimicrobial treatment alone might benefit from this alternative method.[71,73–75]

Conservative therapy with antibiotics alone

This option might be used in patients who are unable to undergo an invasive intervention due to high surgical risk. In the case of complete paresis for >3 days, a surgical intervention is unlikely to improve the neurological deficits. Thus, in these cases, surgery is indicated if antibiotics alone are not sufficient to control the infection. On the other hand, conservative therapy might also be used in patients not suffering from severe loss of spinal cord or cauda equina function. In these cases, frequent evaluation of the neurological status and follow-up laboratory and imaging studies are required. To monitor abscess size, Gd-MRI should be repeated after 2–4 weeks.[78] Referral to a surgical spine centre without delay must be guaranteed if the abscess persists or, even more importantly, if neurological deterioration occurs. In general, non-surgical treatment failure is apparent during the first 48–72 h after the onset of therapy.[6] However, it may also occur at later stages.[44,45,55,76–78]

Duration of antimicrobial treatment

There are no uniform recommendations about treatment duration in SEA, either for the intravenous or the subsequent oral regimen. Furthermore, studies on the bioavailability of antimicrobial agents in the epidural space are lacking. In the literature, the total duration of therapy varies between 4 and 16 weeks, depending on co-morbidities, type of treatment (medical vs. surgical), isolated microorganism, bactericidal effect of the available agent and concomitant spinal infection.[8,9,55] Generally, resolution of the abscess is achieved after 4–6 weeks of treatment.[4] In the case of concomitant vertebral osteomyelitis, treatment is prolonged to 8–12 weeks.[3,5,9,12,48,79] The minimal length of intravenous treatment is not defined, but if concomitant spinal infection is absent, in most studies a treatment duration of at least 3–4 weeks is suggested.[3,12,48,70] Shorter intravenous treatment has only been described in a few cases, and was not associated with treatment failure.[5,77] In the case of concomitant vertebral osteomyelitis, parenteral antibiotics are given for 6–8 weeks.[3,12] It is important to note that intravenous therapy of vertebral osteomyelitis for <4 weeks might lead to treatment failure.[80,81] This may not be true if antimicrobial agents with excellent oral bioavailability can be used. This is the case for quinolones in the case of susceptible Gram-negative organisms, or the combination of quinolones plus rifampin for susceptible staphylococci.[81–83] However, the role of these regimens has not been evaluated in patients with SEA.

Treatment success needs to be confirmed by follow-up imaging studies 4–8 weeks after therapy.[84] The focus should be on soft tissue and not bony findings.[85] Repeating imaging studies at a later stage (e.g. 6 months) is not required if clinical and laboratory investigations are unremarkable.[86]

Key points for clinical practice

The management of SEA should always be multidisciplinary and evaluated urgently. Empiric antimicrobial treatment should include anti-staphylococcal agents and cover streptococci and Gram-negative bacteria. The empiric regimen must be adapted according to regional epidemiological data (MRSA) and predisposing conditions. Based on the size of the abscess and the severity and duration of neurological deficits, the best suitable treatment option (surgical decompression and drainage, CT-guided needle aspiration or antibiotics alone) should be chosen, monitored and repetitively evaluated. Duration of antimicrobial treatment is usually 4–6 weeks for SEA, and 8–12 weeks in the case of concomitant vertebral osteomyelitis.

Outcome

In SEA, two factors are crucial for the assessment of the outcome: mortality and recovery from neurological deficits. Mortality rates range from 2% to 20%; death is usually due to severe sepsis and typically occurs in patients with multiple co-morbidities.[4,5,8,18,55] As with other severe infections, leukocytosis at admission (>14×10^9/l) or

thrombocytopaenia ($<100 \times 10^9$/l) are associated with a worse outcome.[5,14] CRP levels at the time of diagnosis do not predict the outcome. However, CRP levels in the second week after admission can be a prognostic marker.[5]

Other factors associated with poorer outcome include the presence of MRSA, prior spinal surgery, corticosteroid treatment and HIV infection.[3,87]

In a retrospective study including 27 patients with SEA, eight patients had to be re-operated due to a residual or recurrent epidural mass.[29] In these patients, this was associated with the extent of the abscess and the finding of granulation tissue during the first operation.

The final neurological outcome correlates strongly with the severity and duration of neurological deficits prior to surgery.[1,3,4,6,19] Stage III and IV and a duration of more than 24–36 h show the worst recovery rate. Accordingly, post-operative improvement of neurological deficits has been correlated with the rapidity of surgical intervention (within 24 h).[4,8,45] Nevertheless, even timely surgical intervention of patients presenting with neurological deficits does not guarantee complete recovery. Moreover, similar to other causes of spinal cord compression, the time to regain neurological function significantly exceeds the duration until deficits occurred. Therefore, the final neurological outcome should not be evaluated prior to 1 year.[10]

Apart from neurological deficits due to spinal cord compression, the outcome of SEA might also depend on the recovery from concomitant meningitis or encephalitis.

Unfortunately, one-third of patients suffering from SEA still do not have a good outcome, as shown in a recent series.[5,8,14,44] In only 15% of these patients was there no diagnostic delay,[44] indicating that neurological deficits are commonly not recognized and that the urgency for surgical decompression is often not realized. Increased awareness, rapid recognition and quick involvement of a multidisciplinary team remain the key issues in clinical practice. Only then will efficient diagnostic and treatment concepts be assured and outcomes improved.

Key points for clinical practice

The final neurological outcome correlates strongly with the severity and duration of neurological deficits prior to surgery. During the disease course, persistence of an elevated CRP is a prognostic factor. Complete loss of muscle activity (paralysis) lasting longer than 24–36 h is unlikely to improve.

Acknowledgments

We would like to thank Docent Bengt Gårdlund and Professor Jan Andersson (Department of Infectious Diseases, Karolinska University Hospital Huddinge, Stockholm, Sweden) for their critical comments and suggestions. We are indebted to Dr Damien Toia (Department of Radiology and Nuclear Medicine, Cantonal Hospital, Liestal, Switzerland) for providing the images and for helpful discussions.

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