Spinal epidural abscess: the importance of early diagnosis and treatment

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Abstract

Objectives—To remind clinicians of the dangers of delayed diagnosis and the importance of early treatment of spinal epidural abscess.

Methods—A review of the literature on spinal epidural abscess and a comparison of the published literature with local experience.

Results—Imaging with MRI or CT enables early diagnosis of spinal epidural abscess and optimal therapy is surgical evacuation combined with 6–12 weeks (median 8 weeks) of antimicrobial chemotherapy. Clinical features are fever, pain, and focal neurological signs and may be associated with preceding and pre-existing bone or joint disease. The commonest aetiological organism is *S aureus*.

Conclusion—Early diagnosis and appropriate early antimicrobial chemotherapy with surgery is associated with an excellent prognosis.

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Keywords: spinal epidural abscess; diagnosis; treatment; MRI

Spinal epidural abscess is a rare but potentially devastating condition. When recognised and treated early the outcome is excellent.¹ Late diagnosis or inadequate treatment may leave the patient with long term severe or disabling incapacity. The need for early diagnosis and the benefits of appropriate early antimicrobial chemotherapy with surgery are discussed.

Epidemiology

Spinal epidural abscess is an uncommon condition with an estimated incidence of 0.2–2.0/10 000 hospital admissions,^{2 3} and a peak incidence in the sixth and seventh decades of life.^{3 4} At this hospital over a five year period we have seen eight patients admitted from the community with spinal epidural abscess, giving an estimated incidence of 0.25/10 000 admissions. Their median age was 61.5 (range 48–75) The disease is rare in children, with fewer than 90 cases reported.⁵ Conditions commonly associated with spinal epidural abscess include diabetes mellitus, intravenous drug misuse, chronic renal failure, alcoholism, and cancer.^{2–4}

Pathophysiology

Most spinal epidural abscesses are thought to result from the haematogenous spread of bacteria usually from a cutaneous or mucosal

source and this occurred in five of our eight patients. In the series by Baker *et al*,² haematogenous spread of infection was identified in 26% of cases, the sources of bacteraemia including furuncles, pharyngitis, and dental abscesses. Haematogenous spinal epidural abscess are primarily located in the posterior aspect of the spinal canal.⁶ The direct spread of infection into the epidural space from a source adjacent to the spine—for example, spondylitis or discitis—is also well described.^{7 8} In such cases the abscesses are often located in the anterior aspect of the spinal canal and are thought to originate from the adjacent vertebral body. Direct spread from a paraspinal abscess may also occur and was the infective origin in one of our patients. In a recent review of 43 cases of spinal epidural abscess, 44% had coexistent vertebral osteomyelitis.⁹ Vertebral osteomyelitis was present in one of our eight patients (12.5%).

Postoperative abscesses account for 16% of all spinal epidural abscesses² and epidural catheter insertion is another recognised predisposing factor.^{10 11} None of our patients had postoperative abscesses.

Blunt trauma is reported to precede the symptoms of spinal epidural abscess in 15%–35% of cases⁷ and it is postulated that trauma may result in the formation of an epidural haematoma which subsequently becomes infected.¹² A history of recent trauma was recorded in two (25%) of our patients although the source of bacteraemia, as is often the case, was neither obvious nor identified in either patient.

Clinical features

The typical features of spinal epidural abscess are recognisable clinically—notably fever, spinal pain and tenderness, and radiating root pain followed by limb weakness. Pain is the most consistent symptom and occurs in virtually all patients at some time during their illness. Spinal pain and fever are usually the only symptoms present before a precipitous neurological deterioration occurs¹³ and were the only features in two of our patients. Most patients have major neurological signs before surgery³ which are thought to result from a compromised spinal cord vasculature rather than direct cord compression.¹⁴ When septicaemia dominates the clinical picture, as in two of our patients, whose presenting features included jaundice, fever, confusion, and raised fibrinogen degradation products, the neurological symptoms may go unnoticed. This may also be the case in patients confined to bed. In patients with chronic infection,

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The superior quality of MRI over CT is demonstrated in this figure. Sagittal T1 weighted MRI of lumbosacral spine showing osteomyelitis of the L5 vertebral body (large arrow) with spinal epidural abscess L4-S1 anteriorly displacing the meninges (small arrows); (A) before contrast and (B) after contrast. (C) Axial MRI of L5 vertebra showing anterior displacement of the thecal sac by a spinal epidural abscess (arrowed).

constitutional symptoms of fever, weight loss, and systemic upset may predominate over the neurological syndrome¹⁰ and lead to late diagnosis and treatment.

Location

The described sites of spinal epidural abscess are variable. Only 20% occur anterior to the spinal cord.⁴ Our experience suggests that

abscesses in the cervical or upper thoracic region are not uncommon—three of our eight patients had cervical or thoracic spinal epidural abscesses. Larger studies, however, describe a preponderance of lower thoracic and lumbar abscesses.¹⁵ The patients with lumbar abscesses may be misdiagnosed as having a herniated lumbar disc.¹⁶

Microbiology

Staphylococcus aureus was isolated in six of our eight patients (75%). This is the most commonly recognised causative organism and accounts for 57%–73% of reported abscesses.^{2 10 14 17} In a review of five studies between 1930 and 1982,⁴ Danner and Hartman found that *S aureus* was isolated in 62%, other gram positive cocci cultured in 10%, gram negative organisms in 18%, and anaerobes in 2%. After *S aureus*, *Mycobacterium tuberculosis* is the most frequent cause and accounted for 25% of infections in the series reported by Kaufman *et al.*¹⁸ *Streptococcus milleri* has previously been reported as a cause of spinal epidural abscess¹⁹ and is recognised as an organism with a tendency to abscess formation.²⁰ *Haemophilus parainfluenzae*,²¹ *Brucella* species,²² and *Actinomyces israelii*²³ are amongst the many other isolates described. Disseminated fungal infections such as cryptococcosis, aspergillosis, and blastomycosis are rare causes and usually arise in immunocompromised patients.¹⁸ *Aspergillus* species are known to cause spinal epidural abscess in patients with AIDS.²⁴

Imaging

The extent and location of an abscess is best visualised using MRI. Multiplanar imaging and visualisation of the soft tissues—including the spinal cord, paraspinal musculature, and the disc—confers a distinct advantage of MRI over CT and myelography²⁵ and where available, should always be utilised (figure A, B, C).

Antimicrobial therapy

Empirical antimicrobial treatment of spinal epidural abscess should be bactericidal, start early, be delivered intravenously in high doses, and be continued well into the postoperative period.⁷ Initial treatment should be broad spectrum and include a combination of drugs with bactericidal activity against staphylococci, anaerobes, and gram negative organisms. If a methicillin sensitive *S aureus* is isolated, successful treatment is possible with a first generation cephalosporin,⁴ a penicillinase resistant penicillin,²⁶ combination vancomycin plus aminoglycoside,²⁷ or trimethoprim-sulphamethoxazole.²⁸ Parenteral treatment should be continued for at least 4 weeks and may be prolonged for 8 weeks or longer if vertebral osteomyelitis is suspected.^{4 7} When no pathogen is isolated, broad “best guess” bactericidal cover is safest and best.

Surgery

Surgical treatment entails emergency evacuation of the pus with decompression of the spinal cord and nerve roots. In the presence of

paraparesis, the outcome depends on the time that elapses from its onset to surgery.¹⁸ The longer the lapse the greater is the usual long term legacy.

Prognosis

The prognosis is dependent on the clinical and neurological condition of the patient at presentation and any delay in the diagnosis^{4 29} or instigation of appropriate antimicrobial treatment. Patients who present with overt sepsis do poorly. All such patients in one series died.³⁰ Patients who are plegic preoperatively also do poorly, although this is time dependent.²⁹ In the series of Danner and Hartman,⁴ patients with preoperative neurological defects present for less than 36 hours showed some improvement, whereas nine of 11 patients with deficits present for more than 36 hours before surgery did not. In the review by Maslen *et al.*,²⁹ patients paralysed preoperatively for greater than 12 hours did not recover neurological function and those paralysed for greater than 36 hours often died. In our series, the six patients who had developed neurological signs underwent surgery within 8 hours of their onset and had a good outcome. Some authors have found that the outcome of cervical abscesses is worse than those arising in thoracic or lumbar sites.¹⁴ In our series, cervical cases had good outcomes compared with those with thoracolumbar abscesses. Two cervical cases had MRI as the primary spinal investigation and at relatively early stages.

Although the duration of neurological deficit before surgery determines, in part, the neurological outcome, it is not the sole factor. It has been suggested that permanent axonal or cell body damage, for which we do not have a clinical indicator, has prognostic relevance. In our experience one patient who was paraparetic for only three hours before surgery remained paraparetic afterwards. By contrast, another patient who was paraplegic for seven hours made a virtually complete recovery after surgery.

Mortality

Although the mortality from spinal epidural abscess has improved from that described in the early reports, it has remained surprisingly consistent over the past several decades, at about 14%.^{4 29} Mortality is clearly related to delay in instituting appropriate therapy, emphasising the need for early diagnosis and treatment. In addition, these patients should be followed up for several months—even after completion of antibiotic therapy, because of the spinal instability which may result from the infection or the necessary surgical intervention.¹ There were no deaths in our series.

Nutrition

Patients undergoing these procedures often become malnourished and poor nutritional status is associated with increased postoperative complications. Close attention should therefore be paid to the perioperative nutrition and consideration given to preoperative and postoperative nutritional supplementation.³¹

Conclusion

The appreciable neurological recovery seen in some patients reflects the value of prompt diagnosis and early treatment. The key to successful management is early diagnosis, which requires that involved clinicians consider the diagnosis. Repeated spinal and neurological examinations are essential in any patient with an unknown focus of infection and when there is spinal pain or tenderness full investigation is warranted. The increasing availability of MRI may mean that earlier diagnosis and lower morbidity from this condition should become a reality in the near future.

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