Acute spinal epidural abscess without concurrent spondylodiscitis

Successful closed treatment in 10 cases

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We performed a retrospective survey of the clinical records and radiological examinations of 10 patients with a diagnosis of spinal epidural abscess, without spondylodiscitis. All patients had an acute onset of fever and local or radiating back pain. 3 patients had mild, and 1 patient severe neurological symptoms. The diagnosis and subsequent regression of the abscess after treatment were verified by MRI. In all cases, the imaging findings included signs of septic arthritis in an adjoining facet joint. 7/10 abscesses were located in the lumbar region. Blood cultures showed Staphylococcus aureus as the etiological agent in 8/10 patients. In 2 cases, no agent was found, probably due to ongoing antibiotic therapy when the cultures were taken. All patients were treated successfully using antibiotics alone, with complete regression of the neurological symptoms.

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Spinal epidural abscess is a rare disorder, associated with a high risk of neurological sequelae, if diagnosis is delayed (Hlavin et al. 1990, Nussbaum et al. 1992). The exact incidence is not known, but has been estimated at 1–2 per 10,000 hospital admissions (Danner and Hartman 1987, Hlavin et al. 1990). An increasing incidence has also been reported, perhaps due to the increasing number of drug addicts and to more extensive spinal surgery and anesthesia (Nussbaum et al. 1992).

In a large proportion of patients with an epidural abscess, the pathogenesis is due to a direct invasion of the anterior epidural space from a primary infective focus in an adjoining spondylodiscitis (Kaufman et al. 1980, Ericsson et al. 1990, Darouiche et al. 1992, Greenlee 1995). We report our experience with the diagnosis and treatment of patients having an acute spinal epidural abscess without spondylodiscitis.

Methods

We reviewed retrospectively the patient records and the radiological findings in 10 patients having MRI-verified spinal epidural abscess without spondylodiscitis, who had been admitted to the Departments of Orthopaedics and Infectious Diseases at Danderyd Hospital between December 1993 and November 1996.

Results

Clinical data (Table)

All patients had an acute onset of symptoms with fever and pain. In 7 patients, the pain was localized to the back, 2 of them with concurrent radiating pain. 3 patients presented without back pain and had only radiating pain to the scapular region or to a leg. Severe neurological deficits, with pronounced weakness in both arms and a positive Babinski sign were found in 1 patient with a cervical abscess. 3 patients had radicular pain in the lower extremity, 1 of them also had pronounced weakness in one leg. 6 patients had no pathological neurological findings.

Radiology

All patients had been examined with conventional radiographs, 5 patients with bone scintigraphy and 4 with CT of the lumbar spine, without specific findings or suspected infection. Subsequently, all patients underwent a spinal MRI at 0.5 T, including contrast-enhanced sequences. At MRI, no signs of spondylodiscitis were found. Intraspinal findings consisted of expansive inflammatory changes with contrast enhancement in the epidural space. 6 patients had definable cavities. The localization of the abscess was mainly cervical in 1, thoracic in 2 and lumbar in 7 cases. All patients had unilateral inflammatory changes with contrast enhancement and abscess formation in the

Clinical data of 10 patients

Case	A	B	C	D	E	F	G	H	I	K
1	78	f	sk	p	17/168	Th5-L3	L2-3 sin	no	s	4
2	77	f	dm, sk	inf	85/260	L4-S2	L4-S1 sin	no	s	4
3	79	f	mal, sk	s	44/58	C1-Th2	Th1-2 dx	m	s	1
4	70	m		inf	68/169	L1-4	L4-5 dx	rh	u	8
5	81	f	dm, sk	p	94/303	L5-S2	L5-S1 sin	no	s	8
6	71	m	sk	inf	9/183	L1-S1	L3-4 dx	rh	s	7
7	63	f	da, sk	p	17/154	L1-2	L1-2 dx	no	u	15
8	55	m	sk	sp	85/197	L4-S1	L4-5 dx	rh, w	s	3
9	54	m		sp	25/134	Th3-4	Th 3-4 dx	no	s	2
10	27	m	da	sp	44/255	Th6-8	Th6-8 dx	no	s	3

A Age
B Sex
C General health
- da drug addict
- dm diabetes mellitus
- mal malignancy
- sk skin disease/wound

D Suspected diagnosis on admission to the hospital
- s sepsis
- sp spondylitis
- p pyelitis
- inf infection UNS with lumbago

E ESR/CRP on admission (mm/h or mg/L)
F SEA extension
G Facet joint level(s)
H Neurological symptoms
- m compression signs of medulla
- rh rhizopathy, lower extremity
- w weakness, lower extremity

I Agents
- s Staphylococcus aureus
- u not known

K Time (day) duration from first symptom to i.v. antibiotic treatment

dorsal erector spinae musculature. Furthermore, imaging findings suggested septic arthritis in an adjoining facet joint in all patients. The articular findings were contiguous with both the epidural and the paravertebral changes.

Bacteriology

Staphylococcus aureus was the predominant pathogen, isolated in 8 patients by blood cultures. In 2 of these 8 patients, *Staphylococcus aureus* was also isolated from an abscess in the erector spinae and in the psoas muscle, respectively. In the remaining 2 patients, no etiologic agents could be found. In both these patients, blood cultures were drawn after the initiation of antibiotic therapy. In 1 of these patients, an aspiration biopsy from the epidural abscess was performed but yielded no growth, nor did cultures for tuberculosis.

Treatment

The patient with severe upper extremity weakness was given high doses of corticosteroids. All patients were treated with i.v. antibiotics for median 3 (2–8) weeks, followed by oral antibiotics for 5 (1.5–9) months. The 8 patients with *Staphylococcus aureus* in blood cultures were treated with i.v. cloxacillin. The following oral treatment in these patients was flucloxacillin, except in 1 patient treated with clindamycin. The 2 patients with unknown etiology were treated with i.v. cephalosporins for 2 weeks, followed by oral ciprofloxacin.

No epidural abscess was operated on and none of them was drained percutaneously, except in 1 case where an early diagnostic puncture of the epidural space was attempted but yielded no pus and no growth of bacteria. In 2 patients, a percutaneous drainage of an associated erector spinae and psoas abscess was performed.

Clinical outcome

No worsening of neurological symptoms occurred. Complete restitution in all 4 patients with neurological deficits or radicular pain was seen. Good regress of back pain occurred within median 2 (1.5–5) months.

Discussion

To avoid severe neurological deficits, it is important to suspect and detect a spinal epidural abscess as early as possible. The clinical symptoms may be typical, with localized back pain, fever and neurological signs, together with local tenderness on physical examination and elevated inflammatory parameters (Ericsson et al. 1990, Nussbaum et al. 1992). However, in none of our patients was a diagnosis of epidural abscess suspected on admission and in only 3/10 patients was there a suspicion of spondylitis. The marked systemic symptoms and the lack of neurological symptoms in most cases made a correct preliminary diagnosis difficult.

An early diagnosis can be made most accurately with contrast-enhanced MRI. Whereas the findings with plain radiographs and scintigraphy were inconclusive, MRI revealed not only the epidural infection but also evidence of an infection in an adjoining facet joint, as well as in the paraspinal musculature. The presumed pathogenesis in our patients was septicemia, with an acute metastatic infection of the epidural space also involving an adjacent facet joint. This mechanism has previously been considered extremely uncommon and we have been able to find only isolated case reports in the literature (Roberts 1988, Numaguchi et al. 1993, Heenan and Britton 1995, Douvrin et al. 1996, Baltz et al. 1997).

In most reported cases, spondylodiscitis was also present, with extension into the spinal epidural space (Kaufman et al. 1980, Ericsson et al. 1990, Greenlee 1995). *Staphylococcus aureus* was the main pathogen in these cases (Verner and Musher 1985, Danner and Hartman 1987, Ericsson et al. 1990, Hlavin et al. 1990, Nussbaum et al. 1992). It has also been noted that *Staphylococcus aureus* spinal infections are associated with positive blood cultures in nearly every case (Darouiche et al. 1992). It is possible that our patients belong to a somewhat different subgroup. However, the commonest etiological pathogen in our patients also seems to be *Staphylococcus aureus*. The recommended antibiotic treatment of epidural abscesses should at least cover penicillinase-producing *Staphylococcus aureus* and also Gram-negative bacteria and anaerobes, if there is any suspicion of such an etiology or as empiric treatment (Kaufman et al. 1980, Ericsson et al. 1990, Nussbaum et al. 1992, Greenlee 1995).

The long duration of antibiotic treatment in most of our patients (median 6 months) was probably due to the fact that none of the patients underwent neurosurgery and also to uncertainty by the clinician that spondylitis had been excluded. However, even if we take acute spondylitis into consideration, the duration of antibiotic treatment was long. According to the literature, antibiotic treatment for an uncomplicated spinal epidural abscess should be given for 3–4 weeks and 8 weeks, if spondylodiscitis is also present (Verner and Musher 1985, Danner and Hartman 1987, Ericsson et al. 1992, Greenlee 1995). Within 3 months of starting antibiotic treatment, the ESR and CRP values had normalized in all but 1 of our patients and they had no back pain or radicular pain.

Although, to our knowledge, no prospective and randomized study exists comparing nonoperative and operative treatment of spinal epidural abscess, most authors seem to advocate immediate surgical decompression and drainage of the epidural abscess (Danner and Hartman 1987, Peterson et al. 1987, Ericsson et al. 1990, Hlavin et al. 1990, Krauss and McCormick 1992, Nussbaum et al. 1992, Rea et al. 1992). However, in agreement with our observations, a favorable outcome has also been reported without surgical intervention in selected cases (Leys et al. 1985, Mampalam et al. 1989, Baker et al. 1992, Wheeler et al. 1992).

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