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Major complications of epidural analgesia after surgery: results of a six-year survey

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Summary

We performed a retrospective case note review to identify the major complications of epidural analgesia occurring after surgery at our hospital. By cross-referencing the radiology, microbiology and patient information management system databases, we identified patients who had undergone either spinal magnetic resonance imaging or a lumbar puncture within 60 days of surgery in the period from January 2000 to December 2005. Review of these case notes identified six cases of epidural abscess, three of meningitis and three of epidural haematoma. Symptoms of epidural abscess or meningitis developed a median of 5 days after epidural catheter removal. Methicillin-resistant *Staphylococcus aureus* was the predominant pathogen. Epidural haematoma symptoms developed while the epidural catheter was in place. These symptoms were initially attributed to the epidural infusion. Diagnostic delays contributed to adverse neurological outcome in three patients. This study suggests that leg weakness is a critical monitor of spinal cord health. A national database is needed to establish a more accurate estimate of the incidence of major complications and to identify relevant risk factors.

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Epidural analgesia can provide excellent pain relief and may decrease patient morbidity after major surgery [1–3]. However, this technique has significant risks including epidural abscess, meningitis and epidural haematoma. The reported incidence of these complications may be an underestimate, as it is based on case reports [4–11]. In January 2000, we started a survey to determine the incidence of these complications in patients receiving epidural analgesia after surgery in our hospital.

Methods

The survey took place from January 2000 to December 2005 and had Local Research Ethics Committee approval. At the end of each year we gathered data retrospectively from four sources:

1 The acute pain service: to identify all patients receiving epidural analgesia after surgery.
2 Microbiology department: to identify all cerebrospinal fluid (CSF) samples, epidural site swabs or epidural catheter tip specimens.
3 Patient information management system (PiMS): to identify all patients undergoing surgery.
4 Radiology department: to identify all spinal magnetic resonance imaging (MRI) scans.

Each source was asked to provide the patient's surname, forename, hospital number and date of birth, and where relevant the date of the procedure or specimen. The patient information management system also provided details of the operation performed and the surgical speciality.

The data were imported into a database (Access, Microsoft, Seattle, WA). As the acute pain service dataset did not include the hospital number or date of birth, we were unable to identify all patients undergoing investigation within 60 days of epidural analgesia. However, by cross-referencing the PiMS, microbiology and radiology data, we identified those patients who had undergone either a spinal MRI or relevant microbiological investigation within 60 days of surgery. The hospital records of those patients with positive investigations were then reviewed.

Results

Postoperative epidural analgesia was provided to 8100 patients in our hospital between January 2000 and December 2005. We identified six cases of epidural abscess, three of meningitis and three of epidural haematoma.

All patients underwent major surgery. Twelve different anaesthetists sited the epidural catheters. The patients were managed on five different wards after surgery. Two of the complications (both epidural abscesses) followed urgent laparotomy; the remaining surgical procedures were elective. The documented aseptic technique for epidural insertion met recommended standards in all cases [12]. In all patients the epidural space was identified with a 16G Tuohy needle (Portex Ltd, Hythe, UK) using the loss of resistance technique [13]. Three insertions were described as difficult. Transparent non-occlusive dressings were used in the majority of cases to secure the catheter. In January 2004 we changed the epidural infusion regimen from 50-ml syringes containing bupivacaine 0.167% and diamorphine 50 $\mu g.ml^{-1}$ to 500 ml-bags of bupivacaine 0.1% and fentanyl 2 $\mu g.ml^{-1}$ (Table 1).

Risk factors for either infection or bleeding were present in nine patients (Table 2). All patients were given peri-operative thromboprophylaxis and antibiotics for at least 24 h after surgery. Two patients with epidural haematomas were given subcutaneous enoxaparin 20 mg 10 h before and then 9 h after epidural insertion; the other, who was taking clopidogrel up to a week before surgery and then aspirin, was given subcutaneous heparin 5000 IU 1 h after epidural insertion.

Table 1 Details of surgical procedures and epidural techniques. Values are number of patients.

	Abscess/ meningitis *n* = 9	Haematoma *n* = 3
Laparotomy, thoracic epidural	6	3
Joint replacement, lumbar epidural	3	0
Sedated or anaesthetised for insertion		
Sedated	5	0
Anaesthetised	4	3
Loss of resistance		
Air	4	2
Saline	5	1
Difficult insertion		
Yes	2	1
No	7	2
Dressing		
Transparent	5	3
Gauze	4	0
Epidural solution		
50-ml syringe	9	2
500-ml bag	0	1

Table 2 Pre-insertion risk factors for infection and bleeding. Values are number of patients.

	Abscess/ meningitis *n* = 9	Haematoma *n* = 3
Diabetes mellitus	2	1
Malnutrition	2	0
Cancer	1	1
Steroid therapy	1	0
Renal insufficiency	0	0
Pre-operative infection	0	0
Heparin therapy	5	1
Enoxaparin therapy	4	2
Aspirin therapy	0	1

The patients' symptoms at presentation are detailed in Table 3, and relevant clinical timings are shown in Table 4. In eight of the patients with epidural abscess and meningitis, these symptoms appeared after the epidural catheter had been removed. In contrast, the patients with epidural haematomas developed symptoms while the catheter was still in place. Diagnosis was most prompt in the meningitis patients.

The diagnoses were confirmed by MRI scan and/or lumbar puncture except for one patient who had a clinical diagnosis of meningitis made after a negative MRI scan. *Staphylococcus* was identified in eight patients. Methicillin-resistant *Staphylococcus aureus* (MRSA) was the predominant pathogen (Table 5). Four patients underwent

Table 3 Presenting symptoms. Values are number of patients.

	Abscess *n* = 6	Meningitis *n* = 3	Haematoma *n* = 3
Pyrexia	5	3	0
New back pain	6	2	2
Headache	3	3	0
Photophobia	3	2	0
Nuchal rigidity	4	1	0
Leg weakness	1	0	2
Radicular pain	1	0	2

Table 4 Relevant clinical durations. Values are median [range].

	Abscess *n* = 6	Meningitis *n* = 3	Haematoma *n* = 3
Epidural catheter in place; days	5.5 [3–6]	4 [3–4]	3 [2–4]
Time from catheter insertion to onset of symptoms; days	12 [6–31]	5 [4–8]	2 [1–3]
Time from catheter removal to onset of symptoms; days	6.5 [1–25]	1 [0–5]	−1 [−1 to −1]
Time from onset of symptoms to diagnosis; days	2 [0–8]	1 [1–1]	2 [0.75–3]

Table 5 Organisms responsible for epidural abscesses and meningitis. Values are number of patients.

Methicillin-resistant *Staphylococcus aureus* (MRSA)	5
Staphylococcus aureus	2
Coagulase negative *Staphylococcus*	1
Unknown	1

Table 6 Diagnosis, treatment and outcome. Values are number of patients.

	Abscess ***n*** **= 6**	**Meningitis** ***n*** **= 3**	**Haematoma** ***n*** **= 3**
Magnetic resonance imaging scan	6	2	3
Lumbar puncture	1	2	0
Antibiotic therapy	3	3	0
Laminectomy	0	0	1
Laminectomy and antibiotic therapy	3	0	0
Complete recovery	5	3	1
Neurological deficit	1	0	2

surgery; the remainder were given either antibiotic or conservative therapy. Three patients failed to make a full neurological recovery (Table 6).

Discussion

Epidural abscess and meningitis

Epidural abscess and meningitis after epidural catheterisation have reported incidences of approximately 1 : 1000 and 1 : 50 000, respectively [5]. Bacteria may enter the epidural space in a number of ways:

Needle or catheter contamination: barrier precautions [12] were used in all cases after the use of chlorhexidine 0.5% in 70% alcohol for skin disinfection [14–16]. However, surgical facemasks were not documented in all the anaesthetic records as having been used. This may be significant. Published studies have shown that causative organisms have been cultured from both the epidural abscesses and the anaesthetists who inserted the catheter [17, 18]. None of the anaesthetists in our survey underwent subsequent bacteriological investigation.

Epidural solution: all patients had an in-line 0.22 μm bacterial filter present for the duration of the epidural infusion [19]. This filter was not disconnected from the epidural catheter in any of the patients during the infusion period. However, despite the presence of a filter, it has been suggested that frequent syringe changes may be associated with a higher rate of epidural infection [20–22]. We have not identified any further cases of epidural abscess or meningitis since we changed to 500-ml bags of epidural infusion fluid in January 2004.

Superficial infection of the insertion site with subsequent migration along the epidural catheter tract is the usual mechanism whereby bacteria reach the epidural space [4, 23, 24]. The presence of a superficial insertion site infection with a positive microbiological culture was noted in five patients. The insertion site of the other four patients was not commented upon and no swabs were sent from these patients for culture.

A haematogenous source of infection after epidural catheterisation is unusual [4, 23, 24]. None of the patients had clinical evidence of bacteraemia before symptoms of either epidural abscess or meningitis developed, suggesting that a haematogenous source was unlikely.

Predisposing factors for infection: patients who develop epidural abscesses are frequently immunocompromised [4, 5, 9]. Six patients had risk factors for this before insertion; none had evidence of local skin infection at the time of insertion.

Difficult insertion might predispose to epidural abscess by producing either an asymptomatic epidural haematoma [4, 25, 26] or a subcutaneous haematoma [27] that later acts as a nidus for infection. Two insertions were described as difficult. Transparent dressings allow entry site inspection but have been implicated in promoting infection [28]. However, a recent meta-analysis reported no significant difference in intravenous catheter-related sepsis between patients with gauze dressings or transparent dressings [29]. Transparent non-occlusive dressings were used in six patients and gauze dressings in three. The incidence of infection of intravascular and intraventricular lines increases after 3 days [27]. Similarly, most reported cases of epidural abscess have followed epidural analgesia of at least 3 days' duration [9]. Epidural analgesia was continued in our epidural abscess patients for a median of 5.5 days and in the meningitis patients for 4.0 days.

Staphylococcus is the infecting organism usually implicated in epidural abscess formation [4, 5, 9, 30]. *Staphylococcus aureus* was cultured from the insertion site of four patients, from the abscess cavity in two, from both sites in one and from blood in one. One patient did not have any positive cultures despite CSF microscopy confirming meningitis. What is striking about our series is the high incidence of MRSA infections (Table 5). Unfortunately, routine MRSA screening was not performed during our survey. We were thus unable to investigate whether MRSA colonisation of the patient, the staff or the ward predisposed to these complications.

An epidural abscess usually presents about 5 days after epidural insertion with midline back pain and pyrexia [4, 5, 9]. If untreated, neurological injury and paraplegia may develop, usually within a week [4]. Once paraplegia has developed, the prognosis for recovery is poor [4, 31].

Our epidural abscess patients presented a median of 12 days after insertion; in one it took 31 days. As well as back pain, most epidural abscess patients developed headache, photophobia and meningism. However, radicular back pain was uncommon. Clinicians should therefore not rely on this symptom when making a clinical diagnosis. Four epidural abscess patients developed symptoms after discharge from hospital and three of these returned promptly. Unfortunately, the fourth did not return until paraplegic. Meningitis usually follows dural puncture and presents with headache and pyrexia, with only some patients developing nuchal rigidity [5]. Differentiation from postdural puncture headache may not be straightforward [5]. Our meningitis patients presented a median of 5 days after insertion. However, none of them was reported to have suffered a dural puncture during their procedure.

An MRI scan is the investigation of choice for suspected epidural abscess [32, 33]. Delays in diagnosing epidural abscesses are common [4, 5, 9]. The diagnosis was confirmed by an MRI scan in three epidural abscess patients within a day of the onset of symptoms. However, in the other three patients the back pain was ascribed to a musculoskeletal cause and the MRI scan was delayed for up to 8 days. A high index of suspicion is required to minimise diagnostic delays. Meningitis was confirmed within a day of presentation by lumbar puncture in two patients. The other patient had a clinical diagnosis after a negative MRI scan. One epidural abscess patient had both a positive MRI scan and positive CSF microscopy. It is therefore possible that the meningitis patient who did not have a spinal MRI scan had an epidural abscess and not meningitis as was assumed.

A combination of early surgical decompression and prolonged antibiotic therapy is the usual treatment of epidural abscess [26]. However, patients with absent or minimal neurological signs can be managed with antibiotic therapy alone [30]. The neurosurgeons operated on three epidural abscess patients. The others recovered with intravenous antibiotic therapy alone. The patient who returned to hospital paraplegic did not recover neurological function despite undergoing an emergency laminectomy. The remaining patients did not sustain any permanent neurological injury.

Epidural haematoma

Epidural haematomas following epidural catheter insertion are frequently associated with a coagulopathy [31]. The timing of anticoagulant administration is important in decreasing this risk [34, 35]. One patient who suffered an epidural haematoma was given a dose of low molecular weight heparin 9 h after an insertion complicated by the accidental cannulation of an epidural blood vessel. This is contrary to a recent recommendation that low molecular weight heparin administration should be delayed for 24 h if a bloody tap occurs [35], and this may therefore have contributed to the development of the epidural haematoma. Difficulties in identifying the epidural space have been described as a risk factor for the development of a haematoma [36]. One of the insertions was described as difficult because of patient obesity. This patient was given subcutaneous heparin 1 h after epidural insertion. The other two insertions were straightforward. Advanced age, female gender and bony spinal pathology are also reported as being risk factors [5]. Osteoporosis leads to vertebral deformities and spinal canal narrowing [37]. Any collection within the epidural space is thus more likely to compress the spinal cord. The patients with epidural haematomas were all female, their ages ranged from 65 to 79 years (mean = 72 years) and one had a history of lumbar spondylosis.

An epidural haematoma typically presents with radicular back pain, a rapidly progressive sensorimotor deficit and sphincter dysfunction [31]. These symptoms usually develop within 24 h of either epidural insertion or removal, but may be delayed [5]. Two patients initially complained of leg weakness; one of these also had back pain. The other patient developed back pain and bilateral thigh paraesthesia. These symptoms developed a median of 2 days after insertion and in each case while the epidural catheter was in situ.

An MRI scan is the diagnostic investigation of choice. An MRI scan was performed a median of 2 days after the onset of symptoms because in all patients the neurology was attributed to the epidural infusion and the back pain to a musculoskeletal cause. This is a common error [38]. As favourable outcome is dependent on spinal decompression within 8 h of the onset of symptoms, any diagnostic delay is critical [31].

Neurological outcome depends on the severity of the neurological deficit, the size of the epidural haematoma and the time between haematoma formation and surgical intervention [31]. In our patients, the neurological deficits were stable by the time the diagnosis was confirmed. Therefore, only the patient who had pre-existing spinal canal stenosis underwent laminectomy. Neither of the patients who presented with leg weakness made a full neurological recovery. The other patient recovered fully.

Strategy

Patient selection

A recent publication made recommendations for epidural analgesia after surgery, including patient selection [39].

Since January 2000, the number of patients having postoperative epidural analgesia at our hospital has decreased from 1600 to 1200 per year. In orthopaedic surgery, there has been a 60% decrease (from 400 to 150 patients), and in general surgery there has been a 40% decrease (from 700 to 400 patients) despite surgical activity remaining relatively constant. This contrasts with a recent survey that reported an increase in the use of thoracic epidural analgesia in the UK [40]. The selection criteria for epidural analgesia at our hospital appear to have changed. The principal reason for this is presumably an increased awareness of the complications and a move to alternative techniques, e.g. peripheral nerve blocks. However, in the absence of an equally effective alternative, there will continue to be a need for epidural analgesia after major abdominal and thoracic surgery.

Monitoring

Leg strength is the critical monitor of spinal cord health in patients receiving epidural analgesia [41]. Any leg weakness that develops during epidural analgesia must be treated as suspicious until shown to be reversible. The Bromage scale is a simple and commonly used clinical measure of motor block [42]. Since the completion of this survey, we have written an algorithm to clarify the ward management of leg weakness (Appendix). Any patient who has significant leg weakness has their epidural infusion stopped. If there is no recovery of leg strength within 4 h, an urgent MRI scan is performed. Hospitals without a neurosurgical facility need to have clear lines of referral to minimise delays before treatment [33].

Patient information

Epidural abscess may develop after discharge from hospital. All patients receiving epidural analgesia are provided with written information about symptoms of epidural abscess or haematoma along with advice on how and when to seek medical help. Patients who develop superficial infection at the epidural insertion site have this information reinforced during their hospital stay by the acute pain service.

Before we undertook this survey, we knew of seven patients who had suffered a major complication of epidural analgesia. This survey identified a further five. There have also been a number of published reports of epidural abscess and haematoma following epidural analgesia [6–8, 34, 43, 44]. This is therefore not just a local problem. We would strongly recommend that all acute pain services supervising epidural analgesia after surgery perform a regular survey to identify patients who have suffered one of these complications. Such a survey could follow a process similar to ours. The results should then be stored in a national database to provide a more accurate estimate of the risk of these complications. This register might also identify other relevant risk factors, e.g. MRSA infection or colonisation. We understand that the Royal College of Anaesthetists has started a national audit of these complications. We trust that the outcome of this project will be such a register.

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Appendix

Leg weakness and epidural analgesia: a management algorithm