CASE REPORTS

DELAYED PRESENTATION OF AN EXTRADURAL ABSCESS COMPLICATING THORACIC EXTRADURAL ANALGESIA

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SUMMARY
Extradural abscess is a rare but recognized complication of extradural anaesthesia. Previous reports have been associated with a short time interval between extradural catheterization and presentation. We report a patient with rheumatoid arthritis, receiving steroid therapy, in whom an extradural abscess did not present until 23 days after the insertion of a thoracic extradural catheter to provide postoperative analgesia.

KEY WORDS

CASE REPORT
A 62-yr-old retired factory foreman was admitted as an emergency after collapsing while shopping. After a few seconds of unconsciousness, he complained of feeling faint with a constant, dull, non-pleuritic retrosternal and left-sided chest pain. There was no associated dyspnoea or neurological deficit. These symptoms resolved over 30 min. In his past medical history it was notable that he had been admitted to hospital with a myocardial infarction 2 years previously and that for 20 years he had suffered from rheumatoid arthritis. His medication included aspirin 75 mg once a day; penicillamine 125 mg three times daily; flurbiprofen 50 mg three times a day; atenolol 50 mg once a day; isosorbide dinitrate 20 mg three times a day; glyceryl trinitrate as required.

On admission, he was in sinus rhythm with a rate of 50 beat min⁻¹; arterial pressure was 90/50 mm Hg and there was a tender pulsatile midline abdominal mass. Examination was otherwise normal. A syncopal episode related to beta-blockade was diagnosed, with a secondary finding of an abdominal aortic aneurysm. Atenolol was discontinued and isosorbide dinitrate was reduced to 10 mg three times a day. Ultrasound examination confirmed the presence of a 4.8-cm abdominal aortic aneurysm which appeared to have dissected, but not leaked.

Two days after admission, he underwent repair of abdominal aortic aneurysm with an 18-mm straight, non-porous Dacron graft. Before operation, full blood count showed a haemoglobin concentration of 12.3 g dl⁻¹ and a white blood cell count of 4.7 x 10⁹ litre⁻¹. Coagulation screen and serum electrolyte concentrations were normal. Premedication comprised lorazepam 2 mg orally 2 h before induction and hydrocortisone 100 mg i.m. 1 h before induction. Anaesthesia was induced with propofol 160 mg, morphine 10 mg and atracurium 40 mg. Anaesthesia was maintained with infusion of propofol 400 mg h⁻¹, reducing to 300 mg h⁻¹ and atracurium 0.5 mg kg⁻¹ h⁻¹. The trachea was intubated and the lungs ventilated with oxygen and air. Intraoperative monitoring comprised direct arterial pressure measurement, central venous pressure via the left antecubital vein, ECG, end-tidal carbon dioxide, FIO₂ and pulse oximetry.

At induction he was given augmentin 1.2 g and metronidazole 500 mg i.v. as prophylaxis. An extradural catheter was inserted for intra- and postoperative analgesia. The procedure was performed in the operating theatre with the patient in the left lateral position. The skin was cleansed with a solution of 2.5% chlorhexidine in 70% alcohol and a strict aseptic technique was practised.

A Portex 18-guage Mini-Epidural pack was used and the extradural space was identified using "loss of resistance to air". An 18-gauge Tuohy needle was inserted at the T9–T10 interspace and a clear nylon catheter was inserted 5 cm into the extradural space. A bacterial filter (0.2-µm pore size) was attached and the puncture site sprayed with an antibiotic (containing bacitracin, polymyxin and neomycin). The surrounding area was dressed with a plastic, moisture-permeable dressing, impenetrable to bacteria.

An extradural infusion of 0.25% bupivacaine 25 ml, fentanyl (10 ml of a 0.05-mg ml⁻¹ solution) and normal saline 15 ml was commenced at 5 ml h⁻¹ during operation. The effective level of analgesia produced by the extradural infusion during operation could not be determined because the patient was already anaesthetized before insertion of the extradural. However, during surgery there were no clinical indications that the combination of extradural infusion, i.v. morphine given at induction, and the continued infusion of propofol were not effective in producing adequate analgesia. The intraoperative course of the anaesthetic was uneventful. There were
no episodes of hypotension and other measured variables did not differ significantly during the operation. The only anticoagulant used during operation was heparin 5 iu ml⁻¹ in normal saline 150 ml administered locally to prevent thrombosis distal to the aortic graft.

After operation, the extradural infusion was continued for 5 days at a rate of 3–5 ml h⁻¹ but was changed after 24 h to fentanyl (10 ml of a 0.05-mg ml⁻¹ solution in normal saline 40 ml). This regimen provided excellent postoperative analgesia, the infusion rate being based on patient assessment, not-passed frequency and nurse assessment. No abnormality of the extradural catheter site was observed under the transparent dressing during this period. After removal of the catheter, the puncture site was sprayed with a plastic moisture-permeable spray and a dry dressing applied for 48 h. I.v. antibiotic prophylaxis was continued for 48 h after operation. Hydrocortisone 100 mg i.v. four times a day, reducing after 24 h to 50 mg four times a day, was given until the patient was able to tolerate oral medication, when his preoperative regimen was resumed. Postoperative recovery was uneventful and there were no episodes of pyrexia or sepsis; he was discharged home 8 days after operation.

After a symptom-free period at home, the patient was re-admitted to hospital on the 28th day after operation (23 days after removal of the extradural catheter) with a 3-day history of progressively severe, constant, low thoracic back pain associated with parasthesiae and weakness in both legs. Also, he had not passed urine for 24 h. On examination, he had painless retention of urine with tenderness over the lower thoracic spine and signs of a sensory anaesthesia to all modalities below T10. He was afebrile; white cell count was 6.8 × 10⁴ litre⁻¹, with 77% neutrophils.

After urethral catheterization, he was transferred to the neurosurgical unit where a myelogram demonstrated a complete block to upward flow of contrast at the level of T9; computed tomography confirmed that this was caused by a dorsally displaced extradural mass compressing the spinal cord.

An emergency three-level decompressive laminectomy was performed, during which 2 ml of thick pus and a larger volume of granulation tissue was removed. Bacterial culture produced a heavy growth of coagulase positive, penicillin resistant Staphylococcus aureus sensitive to flucloxacillin. I.v. flucloxacillin 2.0 g four times daily and fucidin 500 mg four times daily were started peroperatively and continued for 7 days, followed by 3 weeks of oral flucloxacillin 500 mg four times a day.

Sensory deficit recovered partially, but 1 yr later he was still paraplegic, with an indwelling urethral catheter. Ten months after operation he was found to be neutropenic on routine blood testing and a diagnosis of Felty's syndrome was made.

**DISCUSSION**

The use of an extradural technique has been found to be a safe method of providing postoperative analgesia and neurological problems after its use are extremely rare [1]. Most have been the result of extradural haematoma (usually in patients receiving anticoagulant therapy) [1], and infection of the extradural space is a surprisingly unusual cause of neurological deficit after extradural catheterization. Previous authors have cited diabetes as a risk factor for formation of extradural abscesses [2–4], but we believe that this is the first report of this complication developing in a patient with rheumatoid arthritis receiving steroid therapy.

This is only the second report of delayed presentation of an extradural abscess occurring after extradural anaesthesia. In 1984, McDonogh and Cranney reported a case of an extradural abscess presenting 16 days after removal of an extradural catheter [5]. This case occurred in a previously healthy 72-yr-old man who received 4 days of extradural analgesia after sustaining multiple rib fractures. He became febrile with a chest infection while the extradural catheter was in situ and, although no information was given about blood cultures, the authors suggested that bacteraemia associated with this infection may have been implicated in the formation of an extradural abscess.

Our patient presented with classical progression of symptoms that have been described by both Heusner [6] and Baker and colleagues [7]; these comprise spinal ache, lower limb weakness (with sphincter dysfunction) and, finally, paralysis. However, fever, leukocytosis and neck stiffness that usually accompany acute presentation [6] were not present in our patient. Absence of these symptoms has also been described in patients presenting with chronic extradural abscesses unrelated to extradural anaesthesia [4]. Our patient's delayed presentation 23 days after removal of the catheter made the diagnosis of an infective cause of acute cord compression less obvious, and indeed the diagnosis was not made until laminectomy was performed. Other authors have found that presentation of an extradural abscess after extradural catheterization does not always follow the classical progression of symptoms [5, 8, 9] and this may also lead to delay in diagnosis [4].

In both primary extradural abscess and in those complicating extradural anaesthesia, coagulate positive, penicillin resistant Staphylococcus aureus is the commonest infecting organism. Modes of infection include transcutaneous infection of the extradural space by contaminated needles, injection of contaminated substances from multidose ampoules [9], and possibly haematogenous spread during episodes of bacteraemia [7]. It has not been possible to determine how infection developed in our patient. It is also not certain if the abscess developed acutely or had a more chronic course with symptoms masked by steroid therapy.

It is known that an increased frequency of infection occurs in patients with rheumatoid arthritis and this risk is increased further by steroid therapy [10]. Extradural anaesthesia has been used in patients with rheumatoid arthritis to avoid the risks of spinal cord injury that may accompany tracheal intubation in these patients. This case report suggests that caution should be exercised when this technique is used to provide intraoperative or postoperative...
analgesia in patients with rheumatoid arthritis receiving steroids. This case also demonstrates that, in such patients, early symptoms of an extradural abscess, such as backache, may not develop until weeks after removal of the extradural catheter, yet still progress rapidly to irreversible neurological damage.

In addition to prednisolone, our patient was also receiving two non-steroidal anti-inflammatory drugs (flurbiprofen and aspirin). The effect of such drugs on platelet function may have led to the formation of a small extradural haematoma, and it has been postulated in a previous case report [8] that the presence of a haematoma after extradural catheterization may predispose to infection and abscess formation.

When extradural abscess is suspected, myelography, contrast enhanced computed tomography or magnetic resonance imaging are the diagnostic investigations of choice. Urgent decompressive laminectomy and antibiotic therapy are essential; any delay in surgical intervention or the use of antibiotic therapy alone is associated with a poor outcome [4].

Before this case, our only common contraindication to the use of extradural analgesia had been anticoagulant therapy. However, we would now add to this patients with rheumatoid arthritis receiving steroid therapy.

REFERENCES