EXTRADURAL ABSCESS FOLLOWING LOCAL ANAESTHETIC AND STEROID INJECTION FOR CHRONIC LOW BACK PAIN

C. R. GOUCKE AND P. GRAZIOTTI

SUMMARY

A case is described of extradural abscess following extradural injection of local anaesthetic and steroid for the management of chronic low back pain. The common signs and symptoms are reviewed, possible causes discussed and the association with diabetes stressed.

KEY WORDS


Extradural abscess is an uncommon illness with an incidence of 0.2–1.2 per 10000 hospital admissions [1]. We report the occurrence of an extradural abscess, following extradural injection with local anaesthetic and steroid. As far as we are aware, there has been only one similar previous case report: in that case an extradural abscess followed a single injection of triamcinolone acetonide into the lumbar extradural space [2].

CASE REPORT

A 65-yr-old woman with non-insulin dependent diabetes presented to the Pain Management Clinic with chronic right sided low back pain. The pain was aggravated by movement and radiated to the right hip and thigh. One month earlier, she had been admitted to another hospital where a surgical cervical sympathectomy had been performed for causalgia of the right arm. This was complicated by right phrenic nerve palsy, a staphylococcus (S. aureus) septicaemia and wound abscess.

Examination revealed an obese lady with signs of a peripheral sensory neuropathy affecting both lower limbs. There was tenderness over the lumbar facet joints bilaterally, although facetal compression (lateral flexion and extension) caused no increase in the pain. Straight leg raising was 70° on the left and 60° on the right.

A clinical diagnosis of mechanical low back pain was made, and the patient was prescribed diclofenac 50 mg three times a day, doxepin 10 mg at night and a course of three lumbar extradural injections of local anaesthetic and steroid was arranged to be carried out at 1-week intervals.

Two weeks later, the patient attended for her first lumbar extradural injection, which was performed at the L4–5 interspace with the patient in the sitting position. Using an aseptic technique, the extradural space was identified with loss of resistance to normal saline and 0.25 % plain bupivacaine 6 ml and methylprednisolone 80 mg was injected through an 18-gauge Tuohy needle.

Her pain improved for 2 days, but then recurred. Using a similar technique, a second extradural injection was performed at the L3–4 interspace with 0.25 % plain bupivacaine 8 ml and methylprednisolone 80 mg. There was another good early response, but after 4 days the pain recurred, although not to its initial extent. One week later a third extradural injection was performed in the same manner as the second.

Three weeks later the patient presented to another hospital with left hip, thigh and knee pain following a fall 1 week earlier. She was able to walk only with difficulty and was noted to be tender along the lateral aspect of the left hip, thigh and knee. X-rays of the lumbo-sacral spine showed osteoporosis and old degenerative changes. She was treated with rest and paracetamol with codeine phosphate tablets.

She re-presented the next day with back pain which she claimed had been present for several days. It was sufficiently severe to have required treatment with i.m. pethidine the night before. It did not radiate and there was no associated paresthesiae. On examination, she was afebrile. There was tenderness to palpation over the L2–5 spinous processes. Straight leg raising was 20° on the right and 30° on the left. Knee reflexes were absent, the ankle reflexes normal and the plantar reflexes demonstrated flexion. The bladder was palpable. Lumbar spine x-rays demonstrated no new changes; haematological investigation revealed a haemoglobin concentration of 9.1 g dl⁻¹, white cell count of 15 × 10⁹ litre⁻¹, with 76% neutrophils, and an erythrocyte sedimentation rate (ESR) of 95 mm h⁻¹. Serum creatinine concentration was 249 μmol litre⁻¹ and blood sugar 9.6 mmol litre⁻¹. The patient was observed for 24 h then discharged.

The patient presented again the next day and was admitted with increasing pain in her back, radiating down the left hip and thigh. She was not distressed while lying quietly in bed, but any attempt to examine her caused severe back pain. Her back was tender from L2 to L4, straight leg raising was equal on the left and right, at 45°. Power, sensation and reflexes in the lower limbs were recorded as normal. Her temperature was 38.2 °C.

Investigations now revealed haemoglobin concentration 7.8 g dl⁻¹, white cell count 19 × 10⁹ litre⁻¹, with 93% neutrophils, and toxic granulation with left shift on the blood film. Serum concentration of sodium was 128 mmol litre⁻¹, chloride 84 mmol litre⁻¹, urea 42.8 mmol litre⁻¹, creatinine 286 μmol litre⁻¹ and blood sugar 13.4 mmol litre⁻¹. Liver function tests were marginally abnormal. Urine microscopy revealed a large number of white cells and culture resulted in a growth of Proteus mirabilis of more than 10⁸ bacteria per high power field. Blood cultures grew S. aureus. A bone scan was normal.

Treatment was commenced with i.v. cefotaxime and flucloxacillin and two units of blood were transfused. By the following day she had developed a tender distended bladder, was unable to void urine and the bladder was catheterized. There was little change in her general condition over the next 3–4 days.

Six days after admission a myelogram was performed through an L3–4 dural puncture which yielded purulent cerebrospinal fluid (CSF) with a white cell count of 0.9 × 10⁹ litre⁻¹ and a red cell count of 0.0016 × 10¹² litre⁻¹. S. aureus was cultured from the CSF. The myelogram demonstrated an oedematous cauda equina and decreased CSF space. Post-myelogram computed tomography identified an extradural abscess compressing the spinal cord and occupying most of the spinal canal from L1 to L5.

The patient was transferred to this hospital and underwent urgent decompressive laminectomy. At operation there was an extensive extradural abscess from T12 to L5. The paravertebral muscles from L1 to L5 were inflamed and there was free pus in the interspinous space at L3–4. Laminectomy was performed from T12 to L5, and the pus and extradural fat removed.

The postoperative period was complicated by ventilatory failure requiring prolonged ventilatory support. In the 7th week after operation, the patient was discharged from the intensive care unit. She still had bladder dysfunction requiring continued catheterization and was unable to walk because of marked weakness of both lower limbs. She died 2 weeks later, after deterioration in renal function. Postmortem examination was not performed.

**DISCUSSION**

Extradural abscess is a rare but recognized complication of extradural anaesthesia. Some cases described earlier occurred following continuous caudal anaesthesia, at a time before emphasis on sterile techniques [3, 4]. More recently, extradural abscess has followed extradural catheter insertion [5–8], spinal anaesthesia [9, 10] and infiltration of local anaesthetic into the sacro–coccygeal area [11].

Symptoms from extradural abscess are often subtle [12], with both presentation [13] and diagnosis delayed, sometimes until irreversible neurological signs are present. Important diagnostic features include fever, severe back pain, localized spinal and paraspinal tenderness and leucocytosis. Late signs include nerve root weakness and paraparesis [1, 12]. Myelography and post-myelography computed tomography or magnetic resonance imaging confirm the diagnosis. Treatment involves antibiotics and urgent decompressive laminectomy.

Small haematomata may develop following needle or catheter insertion into the extradural space and this may act as a site for bacterial infection.
growth. Infection may be introduced in several ways. At the time the procedure is performed, if the technique is poor the needle may transport bacteria from the skin to the extradural space. There may be contamination of injected substances, especially if multidose ampoules are used. Spread may occur also from an adjacent locally infected area such as rib, vertebral body or paraspinal muscle. However, the most likely source of infection is haematogenous spread from furunculosis [1]. The commonest organism cultured is *S. aureus*.

In our patient, it is possible that the extradural space was contaminated with *S. aureus* during the septicemia that followed cervical sympathectomy and that presentation of the abscess was merely delayed. However, the operative appearances of liquid pus and inflammation of the paravertebral and interspinous space suggested an acute rather than a chronic abscess.

The controversy surrounding extradural analgesia in febrile obstetric patients was reviewed by Davies and colleagues in 1988 [14]. Bacteraemia occurs in up to 9.7% of these patients, but the incidence of extradural abscess is extremely small and is not increasing, despite the increased use of extradural analgesia during labour, delivery and the postoperative period. This may be because *S. aureus* is rarely involved; indeed, two recently reported cases of bacterial meningitis in parturients after extradural anaesthesia involved streptococci as causative organism [15].

Diabetes mellitus appears to be a risk factor in development of extradural abscess. In two reviews [1,12] involving a total 74 cases of extradural abscess, not associated with extradural injections, 14 patients were identified as having diabetes mellitus. In 1989 Chan [2] reported extradural abscess in a diabetic subject following extradural injection of steroid for sciatica. The association between spinal or paraspinal infection and diabetes has been noted previously and a suggestion was made that if an increased ESR were found in a diabetic patient with backache, specific investigation for spinal or paraspinal infection was warranted [16].

It has been shown that extradural steroids can suppress the hypothalamic–pituitary–adrenocortical axis, which may lead to immunosuppression [17] and this may have been an additional factor contributing to abscess formation in our patient.

This case emphasizes the need for particular caution in diabetic patients and for a high index of suspicion of paraspinal infection when back pain and signs of infection co-exist following extradural injection.

Since this case, we have reviewed our management of diabetic patients and now make a careful examination for occult infection, including measurement of ESR, before performing invasive therapeutic procedures.

**REFERENCES**