Epidural abscess: diagnosis and management

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Introduction
Neurological complications of neuraxial anaesthesia are rare but potentially catastrophic. Major complications include mechanical damage to spinal cord or nerve roots, infection, and spinal cord ischaemia (probably related to prolonged hypotension or administration of vasoconstrictors).1 Epidural abscess is a much feared complication. The reported incidence varies, but is generally very low (up to 0.12%).3 The first written accounts of spontaneous epidural abscess date from 1946 and 1947.4,5 In relation to spinal anaesthesia this complication was first reported in 1974.6

Case Report
A 73 year-old patient with ischaemic heart disease, tricuspid insufficiency, systemic hypertension, hyper-lipoproteinaemia and diabetes mellitus, presented for amputation of a right toe. The amputation resulted in a poorly healing defect, requiring a daily change of dressing. For ischaemic pain at rest and pain relief during the procedure, continuous epidural analgesia was administered. The epidural catheter was inserted at the L4/5 interspace. The catheter was tunnelled 8 cm laterally. Thirty minutes before the change of dressing, a 7 mL epidural bolus was administered of a mixture of 20 ml bupivacaine 0.5%, sufentanil 50 µg and 29 ml of saline. This was followed by a continuous epidural infusion at 5ml/h, which provided good analgesia. After two days the patient was afebrile, but three days after the procedure he reported back pain. On the third day pus was detected around the catheter. The catheter was removed and sent for bacteriological examination. Neurological examination was normal. Antibiotics were commenced in the form of Augmentin 1.2g and Oxacillin 3g IV 8 hourly. The patient was monitored by a neurologist and magnetic resonance imaging (MRI) was recommended should either neurological condition deteriorate or the symptoms persist. After four days he remained afebrile, but in view of a moderate leukocytosis and worsening pain, MRI was performed. The findings strongly suggested an epidural abscess at L2-L4, and within 2 hours, an emergency laminectomy and drainage of abscess was performed. Gentamycin 80 mg IV 8 hourly was added, and also used to rinse the wound postoperatively via inserted drains. Five days later the patient, much improved and without any neurological defect, resumed surgical therapy for his suppurating foot. Staphylococcus aureus was cultured as the pathological organism.

Discussion
Delayed diagnosis of an epidural abscess may cause the death of a patient. Important clinical manifestations include a high fever, back pain and leucocytosis. Due to its rare occurrence, epidural abscess is diagnosed timeously in only about 30% of patients. Clinical symptoms of epidural abscess may be divided into four stages.4,5 Stage 1 is characterised by pain and increased sensitivity in the location of abscess, and high fever. At the same time leucocytosis occurs, accompanied by a raised erythrocyte sedimentation rate. Stage 2 symptoms include radicular pain. Stage 3 is characterised by sensory and motor deficit, and bladder dysfunction. Stage 4 involves gradual paralysis. Forty two cases were described in the English literature between 1974 and 1996.7

This was the first epidural abscess in our unit for the period 2000-2004. During this period we performed 2,246 spinal anaesthetics, inserted 734 epidural catheters for perioperative analgesia, administered corticosteroids and local anaesthetics via the epidural route as a single injection in 507 cases, and inserted 52 tunnelled epidural- and 16 subarachnoid catheters for chronic pain therapy. Patients who had catheters inserted for long term analgesic care, were hospitalised in specialised departments with dedicated and specialised nursing. Epidural abscess is both unpredictable and uncommon.7 There was a good indication for epidural anaesthesia in our case, and strict asepsis was employed during the procedure.

The most frequent symptoms of epidural abscess include back pain (72%), radicular pain (47%), weakness of the extremities (35%), sensory deficit (23%), urinary bladder and gastrointestinal dysfunction (30%) and paralysis (21%).7 Our patient only reported back pain. Moderate leucocytosis and low-grade temperature were common post-surgery, and were not diagnostic in this case. Pus in the area of the catheter did suggest a risk for abscess formation, especially in a diabetic. Dia-

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abetic patients, patients with chronic renal insufficiency, sympathetic dystrophy or immuno-compromise may be at higher risk for epidural abscess formation than the general population, but there is no convincing evidence in the literature that these patients should be denied regional blockade.

Magnetic resonance imaging appears to be the most reliable diagnostic tool available. Computerised tomography and myelography are less reliable. Surgical laminectomy and antibiotic administration is a generally recommended therapeutic method in a case of epidural abscess. Although there are advocates for conservative therapy, most units would opt for surgical intervention, as in this case.

**Conclusion**

Epidural abscess resulting from neuraxial anaesthesia is very rare and at the same time very hazardous. Late or incorrect diagnosis, which occurs in up to 64% of cases, can significantly reduce the chances of complete recovery. Out of seven patients reported to reach the stage of paralysis, five died and two developed permanent paralysis. Of eleven patients reported to suffer neurological deficit, only three patients fully recovered.

Timely diagnosis, aided by the selection of a correct diagnostic procedure, resulted in full recovery of our patient. Despite the rare occurrence of epidural abscess, the risk should not be underestimated, and the clinician should have a high index of suspicion in any case where the diagnosis is a possibility.

**References**


**Figure 1. MRI of Epidural abscess in the lumbar area**

![MRI Image](image-url)