# Acute sacral epidural abscess following local anaesthetic injection

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### Summary

Spinal epidural abscesses are uncommon infections of the central nervous system. Delay in making the diagnosis increases the morbidity and mortality because irreversible neurological damage occurs during this time. We report a 45-year-old male who developed an acute sacral epidural abscess following a local anaesthetic injection given for the relief of low back pain. We believe this is the first documented case of a local anaesthetic injection causing an acute sacral epidural abscess.

KEY WORDS: local anaesthetic, abscess, epidural space.

### Introduction

The incidence of spinal epidural abscesses is 0·2-1·2 per 10,000 hospital admissions (Baker et al., 1975) and most neurosurgical departments treat at least one case a year. The cervical, thoracic and lumbar regions are most commonly involved (Hancock, 1973) and less than 5% of reported cases affect the sacral canal. Most of the latter have been extensions of abscesses in other vertebrae and very few have been confined to the sacral canal. This report presents one such case in a man who had reduced immunity due to undiagnosed Type II diabetes mellitus and was given a local anaesthetic injection into the sacrococcygeal area.

## Case report

A 45-year-old male lorry driver presented with a 4 week history of mild, non-radiating low back pain which was unaffected by coughing or sneezing. On examination, there was tenderness of the sacrum and coccyx but no neurological signs. The duty casualty officer injected approximately 5 ml of 1% plain lignocaine from a sealed ampoule into the sacrococ-

cygeal area producing pain relief. Four days later, the patient returned to casualty complaining of increasingly severe low back pain and was admitted for investigation. There was no history of back injury or recent skin sepsis. His father was an insulin-dependent diabetic.

On examination, he was apyrexial and there was obvious tenderness over the mid-sacrum. Twenty-four hours later, he became pyrexial at 37.6 C and developed the signs of a midline cauda equina lesion; painless urinary retention, loss of sacral sensation (saddle anaesthesia), diminished anal tone and an absent bulbo-cavernosus reflex. There were no other neurological findings.

Investigations: Haemoglobin concentration 11·9 g/dl; while cell count 15·3×10°/litre with 89% neutrophils; erythrocyte sedimentation rate (ESR) 76 mm/hr; fasting blood glucose 10 mmol/litre (normal range 3·3-5·6); serum thyroxine 30 nmol/litre (normal range 45-160); TSH greater than 60 (normal range less than 10). X-rays of pelvis, lumbar and sacral spine were normal. A bone scan was normal. A myelogram showed attenuation and constriction of the subarachnoid space from L5 downwards. Cerebrospinal fluid (CSF) showed no cells, no organisms, protein 1·0 g/litre (normal 0·1-0·5) and glucose 6 mmol/litre.

A laminectomy of the posterior sacral arch and the fifth lumbar vertebra released pus under pressure from an extradural sacral canal abscess. The abscess was drained and culture grew Staphylococcus aureus. Parenteral flucloxacillin was given for one month postoperatively. The diabetes was controlled with oral hypoglycaemic agents and thyroxine replacement started. Six months later, perianal sensation is impaired, but bladder and sexual function are normal.

#### Discussion

Acute epidural abscesses are associated with a characteristic clinical picture. There is a progression of unbearable unrelenting back pain, spinal root pain, impaired cord function with increasing neurological deficit and finally paralysis (Heusner, 1948). The terms extradural and epidural are synonymous. The average time interval between the onset of the pain and paralysis is 4-9 days (Hakin, Burt and Cook, 1979) and the neurological deficit is invariably complete within 48 hr of its onset. There is usually a high fever, tachycardia, leucocytosis and an elevated ESR. CSF analysis shows an elevated protein, a cellular pleocytosis and a normal glucose provided there is no meningitis. Spinal radiography may or may not show vertebral osteomyelitis, but a myelogram is usually abnormal.

Infection of the epidural space arises by either direct extension of a contiguous infection or via the blood from a distant site of infection (Dandy, 1926). The most common organism isolated is Staphylococcus aureus but other causative organisms include Actinomyces israeli, streptococci, Escherichia coli, Pseudomonas aeruginosa, salmonella and anaerobes. In 85% of cases, a primary source of infection is found, usually skin sepsis. In our patient, the local anaesthetic injection probably introduced infection directly into the epidural space at the level of L5-S1. Alternatively, the needle could have entered the sacral canal via the sacral hiatus. Trotter (1947) in an extensive study, found wide anatomical variations in the size, shape and orientation of the sacral bone. The sacral hiatus may be almost closed or widely open depending on the pattern of fusion of the laminae of the sacral arches and sacral spina bifida occurs in about 2% of males and 0.3% females. Thus in some subjects, easy access can be gained to the sacral canal and its epidural space, thereby increasing the danger of random injections in this region.

The incidence of spinal epidural abscess following epidural anaesthesia is surprisingly low considering the number performed. This presumably reflects the strict aseptic technique followed during and after insertion of the catheter. Usubiaga (1975) collected reports of 13 cases, 7 following caudal and 6 after lumbar epidural anaesthesia. North and Brophy (1979) reported two cases, one caused by contamination of the local anaesthetic vial with Staphylococcus aureus and the other, in common with our patient, probably caused by introduction of Staphylococci during insertion of the needle. In 32 patients having

uncomplicated epidural anaesthesia, Barreto (1962) showed growth of bacteria from 3 indwelling catheters at the moment of withdrawal from the epidural space. Moreover, despite strict aseptic techniques, culture of skin swabs were positive in 30% of these patients.

The cause of the 4 week history of low back pain is not certain. We suspected a musculoskeletal cause related to long distance driving and minor degenerative disc disease was evident. We do not think that the pain was caused by a chronic epidural abscess since there was no evidence of osteomyelitis on bone scan or histology and no granulation tissue was found at operation. The hypothyroidism may have delayed and diminished the pyrexia.

Definitive treatment is an urgent decompressive laminectomy and appropriate antibiotics. The excellent outcome in this patient is a result of the early diagnosis and surgical drainage. Surprisingly, there is little evidence the diagnosis is made any earlier today than in the pre-antibiotic era. Local anaesthetic injections given either by themselves or in combination with corticosteroids are commonly used in the treatment of rheumatic disorders (Bourne, 1979). We wish to emphasise the importance of meticulous asepsis when injecting near the spinal canal to prevent introduction of infection and the risk of further epidural abscesses.

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