A severely obese 63-year-old female presented to an outside hospital with a 10-day history of low back pain. On the day of arrival at this institution, she became febrile and confused. She had leukocytosis (20.4 x 10^9/l) and a raised C-reactive proteins (>300). Although she had no meningism, a lumbar puncture was performed which yielded frank pus. Gram staining showed abundant coagulase positive staphylococci. Her obesity precluded initial satisfactory radiological imaging of her spine. Subsequently, the previously normal motor power in her lower extremities deteriorated and MRI demonstrated a spinal epidural collection extending from C3 to L4 vertebral levels (Figures 1 and 2).

The diagnosis was definitively confirmed with an upper thoracic laminectomy (T1-T3) and draining of an epidural abscess. The patient made a steady postoperative recovery, so that 25 days later, at time of discharge from hospital, she was ambulant with assistance and three months later had no discernible neurological deficit.

Spinal epidural abscess (SEA) is an uncommon neurosurgical emergency. Timely diagnosis and management has a direct bearing on outcome. Frequently, early diagnosis is hampered by a low index of suspicion on the part of clinicians due to its nonspecific presentation of localized back pain, fever, neurological deficit and elevated erythrocyte sedimentation rate.1,2 Severe low back pain is cited as the usual initial symptom.1,3 The difficulty in diagnosis has been partly redressed since the introduction of CT and MRI.4-6 Consequently, there has

**Figure 1:** MRI (TR/TE) T1WI Sagittal section depicting the posteriorly located epidural abscess (arrows) abutting upon the posterior aspect of the spinal cord. The abscess extended from C3 to L4.

**Figure 2:** MRI axial section at the T2 level showing the cord pushed anteriorly by the epidural abscess (arrow head). The cord (posterior to the vertebral body) and the abscess have similar signal intensities. The abscess appears to be septated (arrow).
been a significant drop in mortality due to SEA. CT myelography demonstrates extradural compression with block to cerebrospinal fluid (CSF) flow. MRI is now the diagnostic procedure of choice allowing vivid visualization of the anatomical features of the spine. It is particularly suitable for investigating SEA because there is no risk of seeding the subarachnoid space; with gadolinium administration the extent of the abscess cavity is delineated and the degree of thecal compression demonstrated. Furthermore, the risk of causing neurologic deterioration by performing a lumbar puncture below a complete block to CSF flow is eliminated. Depending upon the geographical location, the incidence of SEA is 0.2 to 11.31/10,000 hospital admissions. The variation in incidence is due to population characteristics: areas with burgeoning illicit intravenous drug use and associated problems e.g., AIDS have seen a rise in incidence. Similarly, immunocompromised patients due to other medical conditions are predisposed to SEA. Staphylococcus aureus is the predominant pathogen. Other pathogens include Escherichia coli, Pseudomonas aeruginosa, coagulase-negative staphylococcus, streptococcus, acinetobacter, Mycobacterium tuberculosis, Sporothrix schenckii. The source of SEA remains unknown in about 50% of cases, while hematogenous spread may be demonstrated in the other 50%. Preceding infection at a distant site is common e.g., skin may be the source in 50%. Local extension to the epidural space from an adjacent primary focus e.g., retroperitoneal or mediastinal abscesses, decubitus ulcers and pilonidal cysts occurs in 14% of cases. Spinal epidural abscess may also be iatrogenic e.g., following spinal operations, lumbar punctures, chemonucleolysis, or spinal anesthesia. The recommended treatment consists of surgical decompression combined with the appropriate intravenous antibiotics.

REFERENCES