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CASE REPORT

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Surgical decompression: a life-saving procedure for an extensive spinal epidural abscess

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Abstract Extensive spinal epidural abscesses (SEAs) carry a high mortality rate. Traditionally they are treated non-operatively with long-term antibiotics and/or surgical decompression, but there is a continuing debate as to whether they should be managed by emergency surgical decompression. However, such decisions are made in the light of the clinical setting. We report the successful management of a female patient who presented with features of upper cervical cord compression and later developed septic shock and multisystem failure. Surgical decompression of the cervical spine and irrigation of the epidural space with a paediatric catheter was performed followed by tricortical strut grafting and plating. At review, 36 weeks after surgery, the patient remained asymptomatic, having made full neurological recovery. The purpose of this report is to highlight the importance of emergency surgical intervention for extensive SEA in the presence of progressive neurological loss associated with multisystem failure.

Key words Spinal epidural abscess · Spinal decompression · Spinal irrigation

Introduction

Spinal epidural abscess (SEA) is a rare pathologic entity. Diagnosis may be delayed because of lack of awareness of this condition and its rapid progression. Despite advances in therapy and diagnostic imaging, mortality remains high [1, 9].

The majority of SEAs reported in the literature involve the thoracolumbar region. There are very few reported instances of extensive SEA [9, 13, 15, 17, 18]. We report a case of an extensive SEA extending from C1 to T12 and describe the successful management of this condition.

Case report

A previously fit and healthy 65-year-old woman presented with a 1-week history of bilateral upper limb and neck pain associated with pareasthesia and an unsteady gait. She also complained of shooting pains and paraesthesia down her neck and thoracic spine. There was no history of sphincter disturbances, trauma, malignancy or infection. Her past medical history was unremarkable.

Physical examination revealed an alert afebrile patient in pain with no clinical evidence of anaemia or lymphadenopathy. There was moderate nuchal rigidity and tenderness along the cervicothoracic spine with marked restriction of cervical spine movements. Lhermitte's test was positive.

Cranial nerve examination revealed no abnormalities. Neurological examination of the upper limbs revealed flaccid tone with marked bilateral global weakness (grade 3/5 MRC). The tone in her lower limbs was normal, but motor function was diminished in the hips and knees (grade 4/5 MRC). There was no sensory deficit. The reflexes were absent in the upper limbs, preserved in the lower limbs and the plantars were down going. Rectal examination was normal for sensation and tone.

An initial clinical diagnosis of cervical myelopathy with central cord syndrome was made and the patient was investigated to rule out a compressive cervical cord lesion.

On the evening of her admission, her vital functions rapidly deteriorated. She developed acute respiratory failure with respiratory acidosis and acute hypotension suggestive of upper cervical cord compression involving the cardiorespiratory centres. She was transferred to the intensive care unit (ICU) for ventilatory and inotropic support and monitoring. Laboratory findings showed a leu-

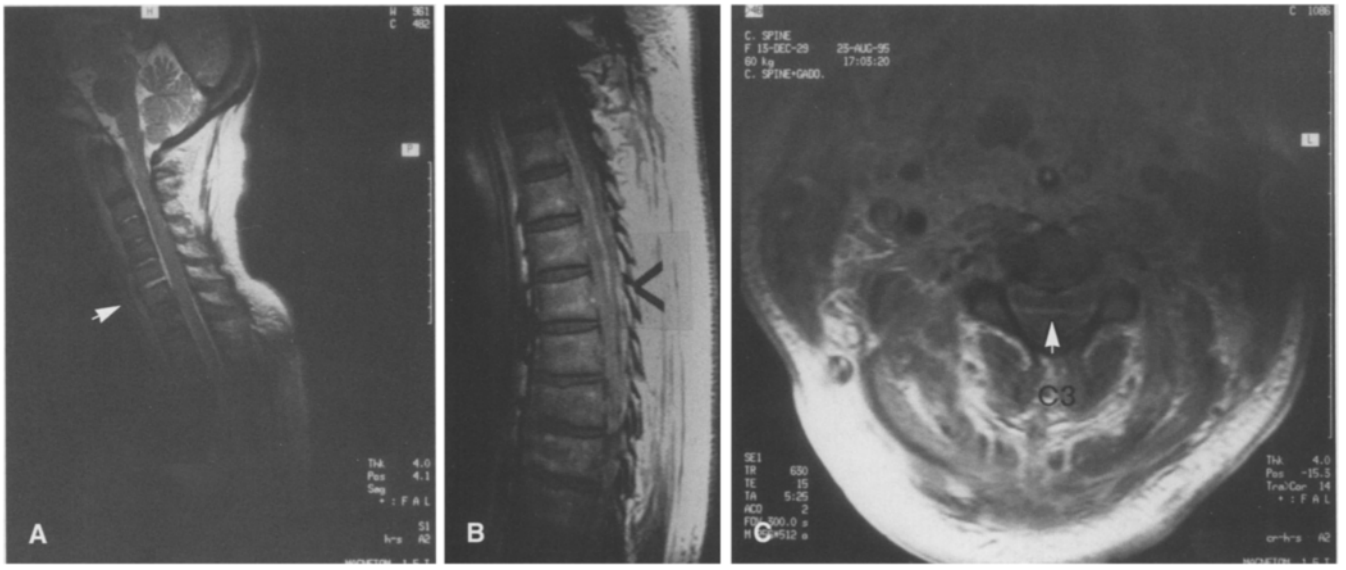

Fig. 1 **A** Sagittal T2-weighted spin echo MR image showing proximal extension of the anteriorly located spinal epidural abscess up to the cranio-cervical junction and evidence of primary discitis at the lower cervical intervertebral discs *(arrowhead)*. **B** Sagittal T1-weighted spin echo MR image showing distal extension of the SEA down to the conus with maximal compression in the mid-thoracic segment *(arrowhead)*. **C** Axial T1-weighted spin echo MR image at C3 showing anterior SEA *(arrowhead)*

cocytosis of 14.4 × 10^9/l, neutrocytosis of 11.72 × 10^9/l and an ESR of > 90 mm/1 h suggestive of underlying sepsis.

MRI of the spine confirmed the presence of spinal infection. The axial sections revealed a peripherally enhancing anterior epidural collection consistent with an SEA. The abscess extended from C1 down to the level of the conus with marked cord compression, particularly at the mid-thoracic level. The abscess was thought to be secondary to primary spondylodiscitis at the lower cervical intervertebral discs, as evidenced on the T2-weighted sequences (Fig. 1).

At surgery the C4-5 and C5-6 discs were bulging and pus was seen draining from the needle entry points in the discs. The dura and cord were oedematous, but cord pulsations were preserved. C5 vertebrectomy was performed and pus and necrotic posterior longitudinal ligament were sent for microscopic examination, culture and sensitivity. A paediatric nasogastric catheter was passed anteriorly, parallel up the cervical and down the thoracic spinal canal, and the epidural space was irrigated with normal saline. Subsequent stabilisation was performed from C4 to C6 with tricortical iliac crest graft and the cervical spine locking plate (CSLP). The patient was returned to ICU for further intensive postoperative monitoring.

Tissue cultures revealed *Staphylococcus aureus* and the patient was therefore treated with appropriate antibiotics. The same organism was also isolated from blood cultures drawn on admission. After a stormy postoperative course, the patient was weaned off the ventilator 1 week after surgery. The patient continued on antibiotics for a total period of 10 weeks.

Serial MRI scans performed 1 month after surgery revealed complete resolution of the epidural abscess and no further evidence of spinal cord compression. Six weeks after surgery the patient had made good neurological recovery and was mobilising well with a Zimmer frame at the time of transfer to a rehabilitation unit. At the last clinic attendance, 36 weeks after admission, the patient had made complete neurological recovery with no symptoms suggestive of a recurrence of the epidural abscess.

Discussion

SEA is a cause of considerable neurological disability. The literature suggests that the incidence has risen from 2.8 to 11.31 cases per 10,000 hospital admissions annually over a 10-year period [8, 13]. *Staphylococcus aureus* is the most common causative organism reported, ranging from 20 to 95% of cases [7, 9, 13].

The specific diagnosis of SEA requires careful evaluation of the history, comorbid conditions, laboratory data and results of imaging studies. Delay in diagnosis is associated with poor outcome [5, 7], and hence MRI is the diagnostic procedure of choice [8, 13]. Hlavin et al. [8] found MRI to be as sensitive as myelography combined with CT scanning, but MRI offers the advantage of being non-invasive.

The management of SEA has remained controversial, with both proponents of non-operative and of operative treatment reporting good results. Several authors have shown satisfactory results in patients treated non-operatively [10, 11, 17, 18]. Leys et al. [10] proposed four indications for the non-surgical management of SEA. These were:

1. Poor surgical candidates
2. Extensive involvement of the vertebral canal
3. Patient not suffering from severe loss of spinal cord or cauda equina function
4. Complete paralysis for more than 3 days

However, non-operative management requires a longer protracted course of antibiotics and close neurological monitoring [3].

On the other hand, several studies have documented abscess progression despite appropriate antibiotic therapy [8, 12]. Hlavin et al. [8] found a significant difference in patient morbidity with non-operative treatment as compared to neurologically normal patients and concluded that early surgical intervention before the onset of neurological deficit is associated with an improved outcome. Others believe that emergency surgery should be performed irrespective of the neurological dysfunction, because disease progression is unpredictable and neurological abnormalities may be irreversible [1, 3].

Surgery is performed with the aim of obtaining a definitive pathological and microbiological diagnosis followed by decompression of neural tissue and, if necessary, stabilisation of the bony elements of the spine. The importance of anterior decompression and posterior stabilisation in diseases anterior to the dura has been emphasised [2, 4, 13]. Other surgical options include laminectomy alone [2, 5, 7] and laminectomy combined with posterior fixation [14]. The choice depends ultimately on the abscess location. Garrido and Rosenwasser [6] reported the use of suction-irrigation technique in order to reduce postoperative infection, while other reports share no such experience.

Although laminectomy is the most common decompressive procedure, there have been reports that laminectomy may worsen the condition and lead to postoperative spinal instability [4, 17, 18].

Our patient did not present with the typical symptoms and signs of SEA. The initial striking feature was that of moderate nuchal rigidity and upper limb flaccid paresis, but no demonstrable clinical sepsis. It was thought that the rapidly expanding epidural abscess resulted in acute cervical cord compression and, with it, compression of the cardiorespiratory centres. In addition, compression of the C3/4/5 motor roots further compromised diaphragmatic function, leading to respiratory failure. This would explain the acute cardiorespiratory failure that necessitated admission to the ICU.

Confirmation of sepsis due to the presence of an epidural abscess was made in the light of the raised C-reactive protein and ESR levels and the MRI study. It can be postulated that the anteriorly situated epidural abscess could expand no further within the spinal canal and was therefore forced out of the neural foramina, thereby causing multiple compression of the corresponding exiting nerve roots. This would explain the flaccid paresis seen on clinical examination. Another possible explanation was acute thrombosis and thrombophlebitis of the epidural vessels, such as that observed by Russel et al. [16].

Our patient developed fulminant sepsis and cardiorespiratory failure, and she suffered from severe loss of spinal cord function as a result of an extensive abscess involving the vertebral canal. She was clearly in a preterminal state and, according to the criteria put forward by Leys et al. [10], would have been managed non-surgically.

Surgery was a technical problem as the extensive anterior abscess could not be adequately drained posteriorly, and the level of operation and approach posed a further dilemma.

Because of the anterior location of the abscess and for ease of surgical approach, we chose to perform a C5 vertebrectomy followed by decompression of the anterior vertebral space with an irrigating paediatric nasogastric catheter threaded proximally and distally. This was followed by tricortical strut grafting and anterior cervical plating of C4 to C6. With continued antibiotic therapy, we report that the surgical procedure and technique of decompression and irrigation have proved successful.

We feel that with the unpredictable and often devastating course of this illness, combined with advances in surgery and postoperative care, early surgical intervention has a definite role in the treatment of such extensive SEA. We advocate that such patients be managed by emergency surgical decompression and stabilisation and recommend that a paediatric catheter can be a very useful device for abscess irrigation and drainage.

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