LESSON OF THE MONTH

Paraplegia with sclerotic vertebral lesions

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Case report

A 53 year old man complained of moderate low back pain associated with loss of 10 kg over six months. There was no history of previous infection. Development of bilateral nerve root pain with leg weakness, with rapid deterioration over several hours precipitated admission. Evaluation at the first local hospital revealed a complete paraplegia and a T11-T12 sensory level. Radiographs of the thoracic and lumbar spine revealed multiple sclerotic lesions of T11, T12 and L5 vertebrae. Prostatic cancer with multiple bone metastases was suspected. The prostate was slightly enlarged and rough. It was decided not to perform surgical decompression, because of the multiple osseous lesions associated with a poor prognosis. However, biopsy of the prostate was normal.

The patient was again referred to our institution a month later with neurological

symptoms. He had no fever. Physical examination showed complete paraplegia. Routine examinations revealed: ESR: 90/h, WBC count: 9000/mm³. Markers of prostatic proliferation were normal. New radiographs showed osteosclerosis of several vertebrae (T11, T12, L1, L2, L5) associated with disc space narrowing of T11-T12, T12-L1 and L1-L2 (fig 1). Magnetic resonance imaging (MRI) showed numerous increased signal intensities in several vertebrae (on T1 and T2 weighted images) with displacement of the spinal cord at the T11-T12 disc space level (fig 2). Vertebral needle biopsies of T11 and T12 vertebrae grew *Staphylococcus aureus*. Blood cultures were negative, but *Staph aureus* was also cultured from the urine. Treatment with pefloxacin and rifampicin was given for three months. Myelography with CT scan revealed a block at the T10 level. Despite the long duration of the paraplegia, laminectomy and debridement was performed at the T10-T11 level. Biopsies taken from adjacent bones and the soft granulation tissue both grew *Staph aureus*. Following the surgical procedure, the neurological deficit was still major and the patient is unable to walk unaided.

Discussion

Spinal epidural abscess (SEA) constitutes one of the most difficult diagnoses in rheuma-

Figure 1 Radiograph of the thoracic and lumbar spine showing multiple sclerotic lesions of T11, T12, L1, L2, L5 vertebrae associated with narrowing of the T11-T12, T12-L1 and L1-L2 disc spaces.

Figure 2 Magnetic resonance imaging showing on T1 sequences abnormal signal intensity of the T11-T12 narrowed disc and adjoining vertebral end-plates associated with compression of the spinal cord (arrow). Multiple signal intensity abnormalities were present from T11 to L3.

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tological practice. This infection, mostly caused by *Staph aureus* and more rarely by Gram negative organisms, commonly affects the lumbar spine.¹⁻⁶ SEA occurs by haematogenous dissemination, or through local extension from osteomyelitis or following spinal procedures.^{1-3 5 6}

The variability of the presenting symptoms often contributes to the lack of clinical suspicion for the condition.^{1 2} In a series of 35 SEA, the initial diagnosis was correct in only seven cases.¹ Commonly, SEA developed in four phases: 1) spinal ache; 2) root pain; 3) progressive involvement of the spinal cord or cauda equina; and finally 4) paraplegia.^{3 7} Absence of clinical and investigational signs in favour of an underlying infection (fever, polynucleosis and inconsistent blood cultures) also contribute to the lack of clinical suspicion.^{1 3 10} Radiographs of the spine may be normal or show radiological signs that could be misinterpreted. In our patient, presence of diffuse osteosclerotic lesions led to the initial suspicion of metastases from a prostatic tumour. However, such bone sclerosis may also be observed in longstanding pyogenic spondylitis, reflecting regenerative changes.¹¹ Furthermore, in our patient, disc space narrowing associated with vertebral lesions was in favour of sepsis rather than metastases.

The diagnosis therefore requires neuro-radiological investigations. Myelography has been the standard radiological method which is still valid.^{5 8} CT scan can only detect loosening of the epidural fat.⁹ At present, MRI is the best procedure for the diagnosis of SEA, showing the epidural mass with a normal or high intensity signal on T1 images and a heterogenous high intensity signal on T2 images.^{2 12-14} Moreover, MRI shows the extent of the SEA^{13 14} and detects associated spondylodiscitis.¹⁵

The most important aspect of the management of SEA is rapidity of diagnosis and treatment. Severe neurological complications could be related to local thrombophlebitis of the epidural veins.^{1 2 4} Paralysis may occur over several hours, independently of the time course of other symptoms.^{4 5 7}

Many authors recommend systematic decompressive laminectomy and operative debridement, followed by one to three months

of antibiotics.²⁻⁴ Neurological postsurgical recovery depends on duration of the initial neurological deficit.^{2 3}

Satisfactory results have been reported recently in patients treated without surgery.^{8 9 13} Leys *et al* recommended four conditions for non-surgical treatment of SEA: poor surgical risk because of severe medical problems; involvement of a considerable length of the spinal canal and extensive epiduritis; complete paralysis for more than three days; absence of significant neurological deficit.⁹ This latest condition is controversial, because of the risk of sudden paraplegia.^{1 2}

The lesson

- Infection of the epidural space can mimic several diagnoses, including vertebral metastases.
- Diagnosis always requires histological and/or bacteriological evidence of infection.
- Delay in diagnosis can result in permanent neurological impairment.

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