Spinal epidural haematoma misdiagnosed as meningitis

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CASE REPORT

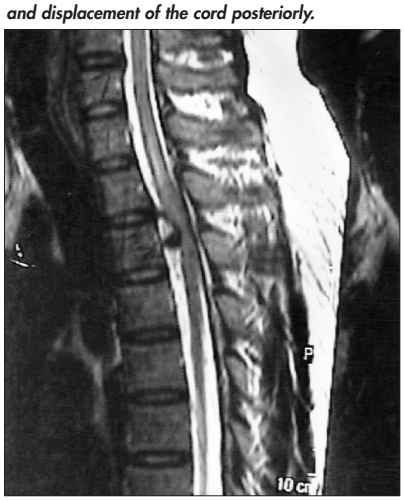
A 32-year-old man developed sudden onset neck pain while at work. There was no headache, fever and no history of head or neck injury. He had been in good health. He continued to work and went home after seeing his doctor, who diagnosed muscle strain. Four days later the pain persisted and he was admitted to hospital. Examination revealed marked neck stiffness, generalized hyperreflexia but with preserved power and a fever of 38°C. Urgent computed tomography of the brain was normal. The blood white cell count was normal. CSF analysis revealed red blood cells and some white blood cells, a blood-stained high protein count of 7 g/litre and a low glucose of 0.7 mmol/litre. Simultaneous blood sugar concentration was 6.7 mmol/litre.

A presumptive diagnosis of meningitis was made and he was started on cefotaxime. Blood and CSF cultures were negative. Magnetic resonance scan of the spinal cord 5 days later showed an acute epidural haematoma at T2–T5 with 40% cord stenosis and cord oedema. He underwent posterior laminectomy for cord decompression; postoperative recovery was uneventful. A subsequent angiogram showed no sign of arteriovenous malformation or tumour and he recovered fully.

DISCUSSION

Spontaneous spinal epidural haematoma (SEH) is uncommon — estimated at one per million patients per year (Groen and Ponssen, 1990). In this patient diagnosis was delayed as meningitis was thought to be the primary diagnosis. Diseases which cause meningeal irritation produce neck stiffness and are life-threatening, e.g. subarachnoid haemorrhage, meningitis, encephalitis, arterial dissection and posterior fossa tumours. A stiff neck can have local causes, including osteomyelitis, abscess and degenerative disease of the cervical spine, and may be seen in patients with Parkinson's disease and oculogyric crises.

Figure 1. Sagittal T2-weighted unenhanced magnetic resonance image of spinal cord showing epidural mass from T2–T5 level with compression and displacement of the cord posteriorly.



Half the reported cases of SEH are related to trauma, bleeding diathesis, anticoagulation and procedural events, e.g. spinal puncture or epidural anaesthesia (Tsai et al, 1975; Boukobza et al, 1994; Rainov et al, 1995). Heavy lifting, coughing and vomiting immediately preceding symptom onset have been reported. The Valsalva manoeuvre is thought to increase pressure in the spinal epidural veins. Vascular malformations, spinal angiomas and rupture of spinal epidural veins have been suggested as possible causes but there is limited pathological documentation in the literature (Scott et al, 1976; Packer and Cummins, 1978). In patients under 40 years of age the lower cervical and upper thoracic cord segments are commonly involved whereas the lower thoracic and lumbrosacral segments are usual sites in older patients. This age-dependent distribution is difficult to explain (Groen and Ponssen, 1990).

Clinical features include neck stiffness, back pain and/or radicular pain with signs and symptoms of spinal cord compression. The investigation of choice is magnetic resonance imaging (MRI) of the spine. Bleeding in the spinal cord produces the pattern of evolution seen with intracranial haemorrhage. Blood products can be visualized on MRI; the age of the clot determines the signal intensity (Fukui et al, 1999). SEH occur close to the bony margins of the spinal canal, usually found dorsally. Subdural haematoma of the spine are even less common and may appear more heterogenous with less well-defined margins than SEH. Other differential diagnoses include subdural or epidural neoplasm or abscess formation.

Decompression laminectomy and evacuation of the haematoma is the treatment of choice unless surgery is contraindicated; Pahapill and Lownie (1998) found mortality was 100% in patients not operated on compared with 26% in those who were. A conservative approach may be considered if there is early, sustained improvement, although this is unusual. The outcome was favourable in this case but delay in treatment of over 36 hours is associated with poor prognosis. Correct diagnosis is important as SEH occurs in young patients (half occur in those under 50 years of age) and it is treatable (Groen and Ponssen, 1990). HM

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