

An unusual case of acute leg weakness

Introduction

Multiple sclerosis is by far the most common cause of non-compressive transverse

Figure 1. Sagittal T2-weighted sequence through the cervical and upper thoracic cord.



myelitis. Other possible causes include infection, sarcoidosis, systemic lupus erythematosus, acute disseminated encephalomyelitis, vitamin B₁₂ deficiency, paraneoplastic syndromes and vaccinations.

Figure 2. Sagittal T1-weighted image through the same level as Figure 1.



Discussion

Syphilitic transverse myelitis is a very rare manifestation of neurosyphilis with only a few cases reported (Lowenstein et al, 1987; Nabatame et al, 1992; Srivastava and Thussu, 2000; Tsui et al, 2002; Matijosis et al, 2006). Studies conclude that syphilitic transverse myelitis is probably a combination of parenchymal infection and inflammation, as well as leptomeningeal arteritis with subsequent cord infarction (Tsui et al, 2002). Magnetic resonance imaging typically shows T2 hyperintense lesions and meningeal enhancement (Tsui et al, 2002). The high intensity areas may indicate ischaemic changes, inflammation

Figure 3. Sagittal T1-weighted post-gadolinium enhanced image. The arrows show meningeal enhancement.



Case Report

A 41-year-old male presented with progressive leg weakness and difficulty with micturition. He had otherwise been well. Clinical examination was normal apart from reduced power (3–4/5) in his lower limbs and a palpable bladder.

The patient was catheterized and had a 1000 ml residual volume. Initial blood tests and magnetic resonance imaging of his brain were normal, but magnetic resonance imaging of the spinal cord revealed extensive abnormality. *Figures 1–3* are selected magnetic resonance images of the cervical and upper thoracic cord.

Figure 1 shows extensive high signal within the cord on T2 weighting, extending from C3 to the upper thoracic region. The cord itself is expanded and the altered signal extended to the T10 level on further imaging. *Figure 3* shows meningeal enhancement (arrows) and this was evident along the whole cord to the conus. There was no compressive lesion. These appearances are consistent with a transverse myelitis.

Lumbar puncture showed an increased CSF white cell count, reduced glucose and increased protein levels. The patient was commenced on intravenous methylprednisolone and cefotaxime. His leg weakness progressed with power falling to 0/5. A sensory level was established at T2. Further blood tests showed a normal serum angiotensin-converting enzyme level, no evidence of tuberculosis, normal autoantibodies and negative viral screen including HIV. Syphilis serology (i.e. venereal disease research laboratory (VDRL), *Treponema pallidum* haemagglutination assay (TPHA) and serum enzyme-linked immunosorbent assay (ELISA)), however, was strongly positive. Further lumbar puncture showed positive CSF antibodies to *Treponema pallidum*. The diagnosis of syphilitic transverse myelitis was made and his antibiotics were changed to intravenous benzylpenicillin. The patient made a full neurological recovery.

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or post-infectious demyelination (Tsui et al, 2002). Signal abnormalities can be present much higher in the cord than is apparent from a clinical sensory level.

Identification of syphilitic transverse myelitis is important as it represents a treatable and potentially reversible cause of acute neurology. Cases previously reported show variable outcomes ranging from complete recovery to more prolonged neurological impairment (Lowenstein et al, 1987; Nabatame et al, 1992; Srivastava and

Thussu, 2000; Tsui et al, 2002; Matijositis et al, 2006). The diagnosis of syphilitic transverse myelitis is based on the signs and symptoms of spinal cord involvement, the characteristic CSF syphilis serology and the magnetic resonance imaging appearances which also exclude a compressive abnormality. Although rare, syphilis should be considered on the differential list of the causes of transverse myelitis. **BJHM**

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