

NEUROLOGICAL PICTURE.....

Clinical MRI dissociation in myelopathy: a clue to sarcoidosis?

A 35 year old man developed, over a one month period, lumbosacral radicular symptoms and prominent nocturnal lower back pain of pulsating–burning quality, together with proximal weakness and an ill defined difficulty in coordination of the lower limbs. General and neurological examination were normal, except for impaired vibratory sensation in the right leg, depressed left knee reflex, and bilateral dysesthesias with L3–L4 distribution. Despite the patient's complaints, gait and strength were not impaired.

Haematochemical investigations, including autoantibody search, were negative. MRI revealed a diffuse intramedullary process, extending from C1 to the conus medullaris. CSF examination revealed mildly increased albumin (6.7 g/dL), IgG (11.7 mg/dL), and lymph monocytes (6.4 cells/mm³), without intrathecal IgG synthesis. CSF infections (herpes simplex virus 1 and 2, varicella zoster virus, cytomegalovirus, and influenza) were ruled out by either PCR or cultures (including microbacteria). Neurophysiological investigations (somatosensory evoked potentials (SSEP) and motor evoked potentials, electromyography/electroneurography) were normal except for delayed cortical response on right tibial SSEP. Chest radiographs revealed bilateral hilar opacities, consistent with a lymphadenopathy, while thorax CT showed mediastinal and hilar enlarged lymph nodes, without pulmonary infiltrates.

At this time, laboratory findings of increased serum angiotensin converting enzyme (30.9 U/L, normal range: 8–21.4), supplied an otherwise unpredicted answer. A mediastinoscopic lymph node biopsy showed noncaseous granulomata consistent with sarcoidosis. Oral prednisone 75 mg daily and intramuscular methotrexate 10 mg weekly were started. One month later, following clinical and radiological improvement, steroid was gradually tapered.

In contrast to previously reported cases, and similar to what can be observed in lung sarcoidosis, in spinal cord disease the radiological abnormalities are more striking than the clinical manifestations. This should be borne in



Figure 1 MRI of the spinal cord, cervical tract, sagittal image. (A) Sagittal T2 fluid attenuated inversion recovery (FLAIR) image (RT 6000-ET 120), showing expansion of the cord at the cervical level and a diffuse abnormal signal involving the entire medulla. (B) T1-Weighted sagittal image obtained after the administration of gadolinium-DTPA (RT 500-ET 11), showing patchy enhancement extending to several levels, with focal areas of more intense enhancement. There is no epidural collection of inflammatory tissue or abnormal enhancement around the cord.

mind when sarcoidosis is suspected. The available literature on sarcoid myelo-



Figure 2 MRI of the spinal cord, dorsal tract, axial image. Axial T1-weighted gadolinium enhanced image (D11 level), showing intramedullary disease sparing the nerve roots and the surrounding meninges.

pathies does not fully elucidate whether correlations between neurological symptoms and MRI abnormalities are of diagnostic value, or bear prognostic implications. Investigations are often discontinued once tissue diagnosis is achieved, leading to incomplete data collection. This understandable tendency has limited our knowledge on the usefulness of non-invasive methods for investigating neurosarcoidosis. We believe that sarcoidosis should be considered in the differential diagnosis of a myelopathy, especially when paucisymptomatic, in order to avoid diagnostic delay in this steroid sensitive condition. Extraneurological manifestations, when searched for, are almost invariably present, even though clinically silent,¹ while delayed diagnosis and invasive procedures may lead to persistence of the medullary insult and thus to permanent disability.^{2,3}

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