

Pilocytic astrocytoma of the spinal cord in an adult

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Case presentation

A 42-year-old man presented with a 9-month history of progressive numbness and paresthesias involving the upper limbs. His symptoms started at the fourth and fifth digits of both arms, and ascended to involve the elbows. Within several weeks, the lower limbs became involved with the numbness and pain starting at the toes and progressing up to mid thorax. He had no bowel or bladder involvement. His examination showed no stigmata of systemic disease. He had minimal weakness at hip flexion, upper motor neuron signs in all limbs, decreased sensation in C8 distribution bilaterally, and a sensory level at T4 sparing proprioception. Magnetic resonance imaging (MRI) of the spinal cord 6 and 9 months (September 2006, December 2006) after symptom onset

revealed T2 prolongation and cord edema (Fig. 1a, c) with an 8 mm enhancing intramedullary lesion at the C5-6 (Fig. 1b, d). The possibility of demyelinating disease was considered. Thoracic and brain MRI were unremarkable. Further workup included cerebrospinal fluid (CSF) which revealed leukocytosis of 13 with 88% lymphocytic predominance, protein of 310 mg/dl, normal glucose and two oligoclonal bands not present in a corresponding serum sample. The patient continued to develop progressive neurological deficits that did not respond to treatment with 5 days of 1 g intravenous Methylprednisolone. Repeated CSF exam showed persistence of protein elevation (289 mg/dl), normalization of leukocytosis and absence of oligoclonal bands. Both examinations showed normal cytology. Anti nuclear antibodies (ANA), Lyme serology, angiotensin converting enzyme (ACE), HTLV-1 serology, B12 level, anti-neutrophilic cytoplasmic antibodies (ANCA) were all normal. CT scans of the chest, abdomen and pelvis excluded occult malignancy and lymphadenopathy. Magnetic resonance angiography (MRA) and spinal angiogram showed normal cerebral and spinal vasculature. Repeated spine MRI (February 2007, July 2007) showed mildly increasing edema at the site of cervical spinal cord lesion, but stable lesion size and unchanged enhancement pattern. Differential diagnosis included a neoplastic process due to the persistent duration of enhancement, the elevated protein in the CSF, and absence of clinical or radiographic improvement after treatment with intravenous steroids.

As the etiology of the lesion was not clear preoperatively, i.e. intramedullary neoplasm or ischemia from stenosis, the patient underwent C4-T1 decompression, posterior spinal fusion, posterior laminectomy, and intradural subtotal mass resection. Intraoperatively the surface of the cord and the vasculature looked normal. The cord

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