



Case report

Primary spinal oligoastrocytoma mimicking longitudinally extensive transverse myelitis

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ABSTRACT

Longitudinally extensive transverse myelitis (LETM) is most commonly associated with neuromyelitis optica spectrum disorders (NMOSD). However, a wide range of etiologies may produce longitudinally extensive spinal cord lesions (LESCLs) on imaging. We highlight the case of a patient with a spinal cord tumor whose imaging showed LESCL and was diagnosed with LETM. He did not respond to immunosuppression and subsequently developed a progressive and protracted clinical course. Thoracic cord biopsy performed 6 years after symptom onset showed primary spinal oligoastrocytoma. We discuss the features that should raise suspicion of a neoplasm in the context of LESCL and serve a reminder that not all LESCLs are inflammatory.

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1. Introduction

Longitudinally extensive transverse myelitis (LETM) is defined as an intramedullary lesion extending over 3 or more vertebral segments in the context of suspected inflammatory spinal cord syndrome (Kitley et al., 2012). It is most commonly associated with neuromyelitis optica spectrum disorders (NMOSD) and is an important feature of the 2015 international consensus diagnostic criteria for neuromyelitis optica spectrum disorders (Wingerchuk et al., 2015). However, longitudinally extensive spinal cord lesions (LESCLs) can not only be seen in immune mediated inflammatory-demyelinating conditions, but also in a diversity of pathologies that include infectious, vascular, neoplastic, paraneoplastic and metabolic causes (Kitley et al., 2012). These etiologies need to be carefully considered in a patient presenting with LESCL.

2. Case report

A 51-year-old Chinese male presented to another hospital in July 2008 with gradual onset bilateral thigh numbness and weakness over a few months. Magnetic resonance imaging (MRI) of the

thoracic spine showed a non-enhancing T2-hyperintense lesion from T8 to T12 with cord expansion (Fig. 1A). Syphilis serologies, anti-double stranded DNA antibody, complement levels, anti-Ro antibody, anti-La antibody, anti-Smith antibody, anti-ribonucleoprotein antibody, anti-topoisomerase 1 antibody and erythrocyte sedimentation rate were normal. Lumbar puncture was not performed and he was not treated with immunosuppression.

He presented to our hospital in August 2009 with progressive lower limb weakness, erectile dysfunction and urinary retention. Examination revealed spastic paraparesis and sensory level at T12. MRI spine showed a T2-hyperintense lesion from T6 to T12 with patchy contrast enhancement and cord expansion (Fig. 1B–E). Axial imaging showed it occupying nearly the entire cross-section of the cord at T8 with subtle contrast enhancement (Fig. 1F and G). MRI brain was normal. Cerebrospinal fluid (CSF) analysis showed 6 cells/ μ L, protein level of 41 mg/dL and oligoclonal bands negative. Serum aquaporin-4 (AQP4) antibody, human immunodeficiency virus serology, anti-nuclear antibody, anti-cardiolipin immunoglobulins and anti-neutrophil cytoplasmic antibody were negative. He was diagnosed with LETM and treated with pulsed intravenous (IV) methylprednisolone 1 g daily for 5 days followed by oral prednisolone taper, with symptomatic and objective improvement. He was able to ambulate independently, but this improvement plateaued after one month.

He remained stable till February 2010 when he noted worsening lower limb weakness. Further pulsed IV methylprednisolone did not provide improvement and he required ambulatory

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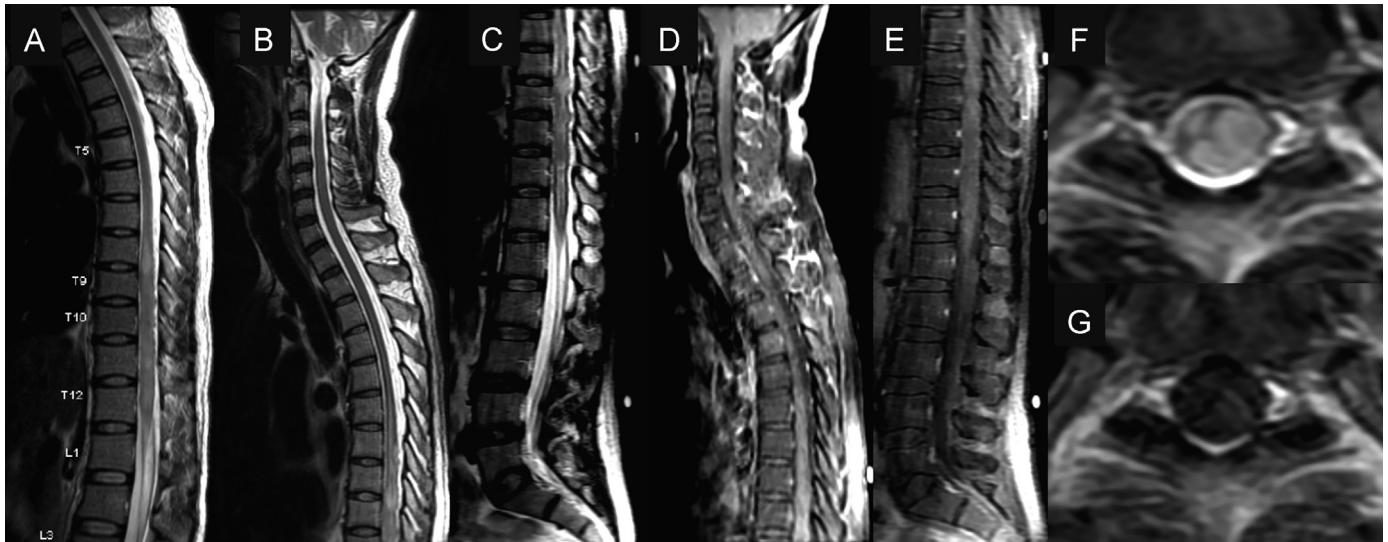


Fig. 1. Serial MRI spine from 2008 to 2009. (A) MRI spine showing T2-hyperintense lesion from T8 to T12 with cord expansion in 2008. (B, C) Rostral extension of T2-hyperintense lesion to T6 with cord expansion and (D, E) subtle patchy contrast enhancement in 2009. (F) Axial imaging showing the lesion occupying nearly the entire cross-section of the cord at T8 with (G) subtle contrast enhancement in 2009.

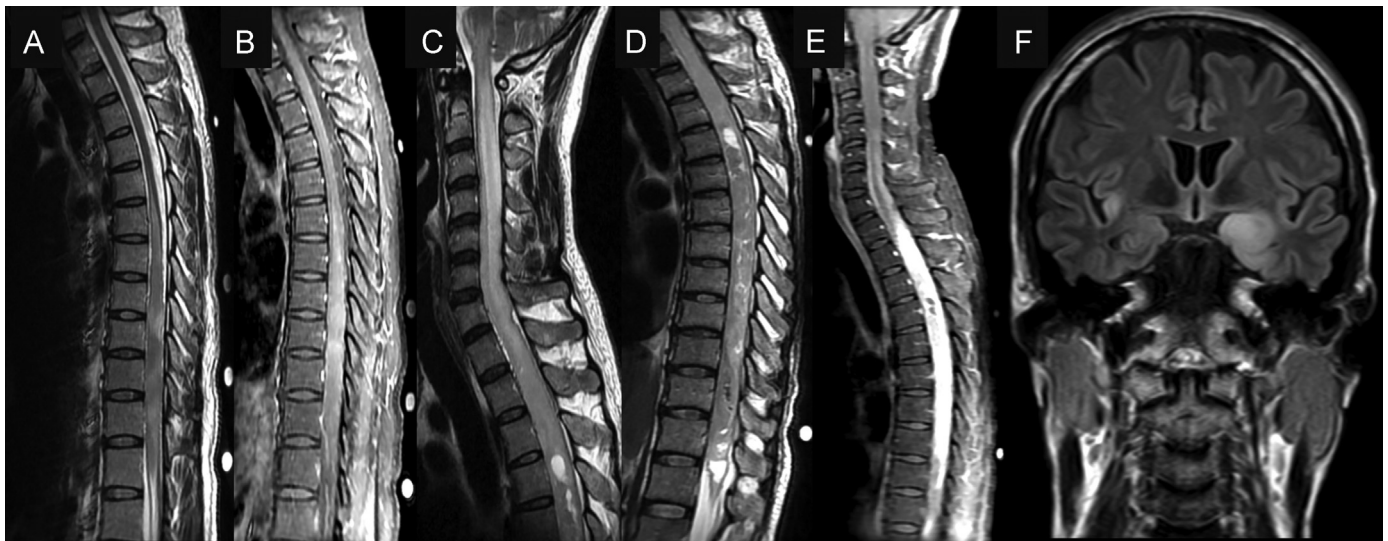


Fig. 2. Serial MRI spine and brain from 2010 to 2014. (A) Slight rostral extension of T2-hyperintense lesion to T5/6 junction with increased cord expansion and (B) increased heterogeneous contrast enhancement in 2010. (C, D) Extensive T2-hyperintense lesion from cervico-medullary junction to conus with marked cord expansion, cystic changes and (E) avid contrast enhancement in 2014. (F) MRI brain showing FLAIR abnormalities in the left medial temporal lobe, left caudate nucleus and right insular region in 2014.

assistance by September 2010. Radiological progression was noted with increased cord swelling and contrast enhancement (Fig. 2A and B). There was no response to additional pulsed steroids and 5 doses of monthly IV cyclophosphamide (15 mg/kg). He declined plasma exchange. In January 2011, his sensory level had progressed up to T8 and he required intermittent urinary catheterization. Repeat MRI spine showed stable findings. He declined further treatment with Rituximab as well as surveillance imaging. In 2012, he had become wheelchair-bound and subsequently developed recurrent urinary tract infections and pressure sores.

In February 2014, he developed gradual onset bilateral upper limb weakness. Examination revealed weakness of C7, C8 and T1 segments with complete paralysis of his lower limbs. MRI spine showed a T2-hyperintense, enhancing, cervico-medullary junction to conus lesion with marked cord expansion and cystic changes (Fig. 2C–E). In view of the clinical progression and neuroimaging findings, neoplasm was considered. Histology of cord biopsy performed at T10 confirmed World Health Organization (WHO) grade

III oligoastrocytoma (Fig. 3). Fluorescent in-situ hybridization analysis of 1p19q chromosomal co-deletion was negative. MRI brain showed intracranial involvement with multiple areas of fluid attenuated inversion recovery (FLAIR) abnormalities (Fig. 2F). Palliative chemotherapy with temozolomide was commenced without clinical or radiological improvement.

3. Discussion

The distinction between inflammatory and neoplastic causes of LESCL can be challenging with intramedullary spinal cord tumor (IMST) mimicking myelitis and vice versa. Jacob et al. (2006) reported a case of cervical cord ependymoma that was mistaken as relapsing myelitis because of positive CSF oligoclonal bands and initial response to steroids. Neoplasm was suspected after the patient developed severe neck pain despite incremental steroids. Conversely, Brinar et al. (2006) reported 5 cases of inflammatory

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