CLINICAL VIGNETTE

Emergency Department Presentation of Idiopathic Transverse Myelitis

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A 65-year-old female with past medical history only significant for hypertension, presented to the emergency department with complaints of numbness. She was in her usual state of health until 5 days prior to admission when she noted sudden onset numbness from the groin and lower abdomen, extending to the lower mid chest region and upper back. Over the next 2 days prior to her emergency department visit, symptoms progressed to involve her bilateral lower extremities and left hand. The patient also described a sensation of tingling and electrical shocks in these regions. On the day of admission, she complained of left hand weakness in addition to progressively worsening numbness. She denied headaches, dizziness, dysarthria, dysphagia, visual changes or ataxia. She denied bowel or bladder incontinence. She also denied a personal or family history of demyelinating disorders such as multiple sclerosis.

On physical exam, the patient was in no distress but seemed slightly anxious. On neurological exam, cranial nerves were intact, extra-ocular and pupillary exams were normal. Sensory exam was normal and symmetric to light touch and pinprick in all areas of subjective complaints. She had dysmetria with left hand movements and mild left upper extremity weakness with noticeable handgrip weakness. The rest of the neurological exam was essentially normal.

Based on the patient's complaints and examination, MRIs of the brain, cervical spine and thoracic spine were performed and she was found to have C2-C4 intramedullarly enhancing lesion with surrounding edema (see figure 1). MRI of the brain was unremarkable. A lumbar puncture showed mild monocytic pleocytosis with WBC=36. CSF glucose was normal, with slightly elevated protein. A multiple sclerosis panel, neuromyelitis optica (NMO) antibody, HIV, HTLV1, VDRL and test for autoimmune disorders were all negative.

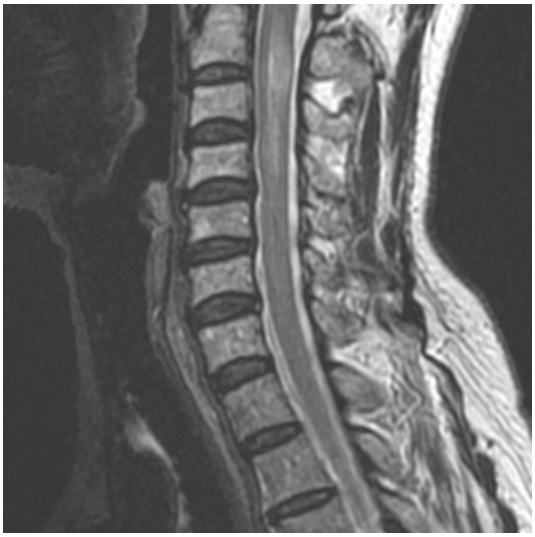

Figure 1: Enhancing lesion within the spinal cord with surrounding edema from C2 to C4.

The suspected diagnosis was idiopathic transverse myelitis. She was started on high dose methyl-prednisolone for 5 days followed by a prednisone taper. Her symptoms improved significantly with near-resolution after 3 days of treatment. A follow-up cervical MRI, a month after her diagnosis, showed a marked decreased size of the T2 signal abnormality in the cervical spine without enhancement.

Transverse myelitis is an immune-mediated process that causes neural injury to the spinal cord leading to variable degrees of weakness, sensory defects and autonomic dysfunction. Transverse myelitis may be idiopathic[1, 2] or exist as part of CNS disease such as multiple sclerosis or multi-systemic disease such as systemic lupus erythematosus. The incidence of transverse myelitis ranges from 1-8 cases per million per year[3].

Diagnostic criteria for idiopathic transverse myelitis were proposed by the Transverse Myelitis Consortium Working Group

(TMCWG). These include bilateral signs or symptoms of spinal cord dysfunction, a defined sensory level, inflammation within the spinal cord demonstrated by CSF pleocytosis, elevated IgG index, gadolinium enhancement, and progression over four hours to about twenty-one days[1].

Patients may have preceding non-specific symptoms of fever, nausea and myalgia[4]. These prodromal symptoms are seen in 40% of pediatric patients 3 weeks prior to onset of transverse myelitis. The pathogenesis of transverse myelitis is suspected to be immune-mediated[5]. This is supported by the presence of CSF pleocytosis and the breakdown of the blood-brain barrier within a localized area of the spinal cord.

Important differential diagnoses to exclude in patients with neurological complaints such as in this report include cord compression from a mass, spinal cord infarct, an epidural abscess, epidural hematoma, and central disc herniation

Intravenous steroids are the treatment of choice for transverse myelitis[6,7]. Although there are no randomized controlled studies, clinical experience with treatment of related disorders such as multiple sclerosis and spinal cord injuries supports this therapy. Another treatment used for severe transverse myelitis unresponsive to steroids is plasma exchange[8]. Patients felt to benefit from plasma exchange include those not responding to high dose steroids after 5-7 days, patients with inability to ambulate and those with severe autonomic dysfunction.

Transverse myelitis patients exhibit variable prognosis. Some may recover neurological function without therapy within 6 months of diagnosis[9]. Poor outcomes are associated with patients who present with back pain as initial complaint, rapid progression over a few hours, spinal shock, and sensory disturbances involving the cervical segments of the spinal cord[10].

Transverse myelitis is a rare disorder that can be difficult to diagnose in the emergency department because of the variable presentation. It is important for the emergency physician to include this differential diagnosis in the evaluation of non-specific sensory deficits. Early diagnosis and treatment may alleviate the accompanying morbidity and mortality seen with transverse myelitis.

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