# A rare cause of myelopathy in a young female

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A 35-year-old female patient presented with a 2-week history of progressive numbness and burning sensations starting in both lower limbs and ascending upwards to the trunk. There was no history of weakness or bladder involvement. There was a history of fever 1 week before symptom onset. There was no history of headache, vomiting, joint pains, or skin rash.

General examination showed no significant findings. The patient was afebrile with a pulse rate of 74/min and blood pressure of 130/80 mmHg. Clinical examination revealed loss of sensations over both lower limbs and trunk from the T4 dermatome downward. Cranial nerves and motor power were normal. There were no meningeal signs. Deep tendon reflexes in both lower limbs were exaggerated with extensor plantars bilaterally. A clinical diagnosis of cervical myelopathy was made. The differential diagnosis for subacute myelopathy included post-infectious demyelination, vasculitis syndromes, infections, degenerative disc disease, nutritional myelopathies, and spinal cord tumors.

Blood investigations revealed strongly positive serum myelin oligodendrocyte glycoprotein (MOG) antibody. Magnetic resonance imaging (MRI) of the spine showed a long segment of hyperintensity in the cervical spinal cord extending over C5–7 segments with contrast enhancement (Figs. 1 and 2). Brain MRI was normal (Fig. 2). Cerebrospinal fluid examination showed no infection and oligoclonal bands were negative. Serum for Vitamin B12 levels was normal and vasculitic markers were negative.

She was started on pulse dose intravenous steroids methylprednisolone 1000 mg daily for 5 days followed by a tapering schedule of oral steroids prednisolone, initially at 1 mg/kg dose and tapered slowly over 8 weeks. She had good improvement in symptoms and is on regular follow-up. Repeat imaging after 3 months showed a complete disappearance of the cord lesion (Fig. 3). In case of recurrence, she is planned for immune modulation with mycophenolate or rituximab.

MOG antibody disease is an autoimmune disease that causes inflammation of the central nervous system [1]. It is caused by the immune system attacking the MOG protein, which is found on the myelin sheath of nerves [2]. The median age of onset is usually the fourth decade of life, with optic neuritis being the most frequent presenting symptom. Other presentations include a transverse myelitis and encephalitis-like presentation. There is no precipitating factor in most of these cases but viral infections with herpes simplex and also Epstein Barr viruses are reported. Disease course can be either monophasic or relapsing, with subsequent relapses most commonly involving the optic nerve and spinal cord [3]. Transverse myelitis occurs most commonly in the cervical and thoracic spinal cord [4]. Lesions in the brain and spine can be identified using MRI sequences. Brain lesions are usually bilateral, poorly demarcated, and can involve the brainstem regions [5]. Contrast enhancement is usually seen in most cases.

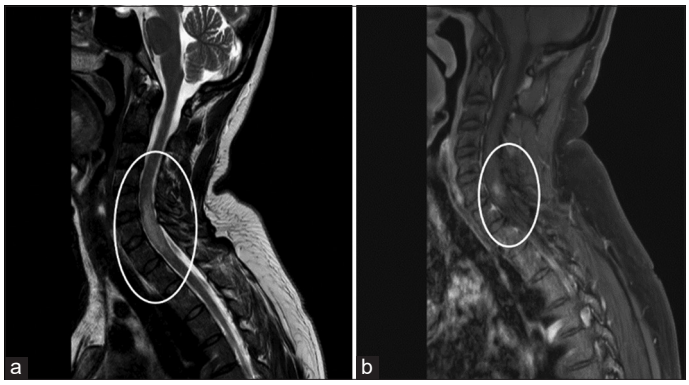


**Figure 1: Magnetic resonance imaging spine sagittal section T2 sequences showed a long segment of hyperintensity (a) in the cervical spinal cord with contrast enhancement (b)**

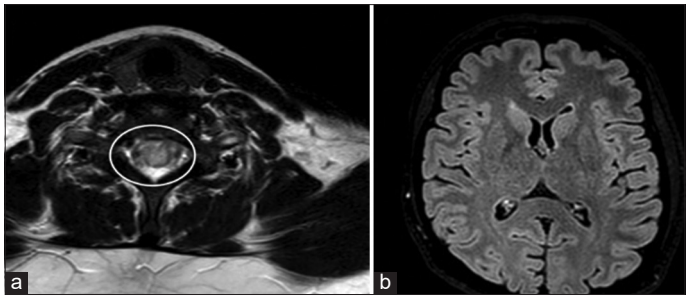


**Figure 2: Magnetic resonance imaging cervical spine T2 axial sections showed cord hyperintensity (a) while brain T2 axial section was normal (b)**

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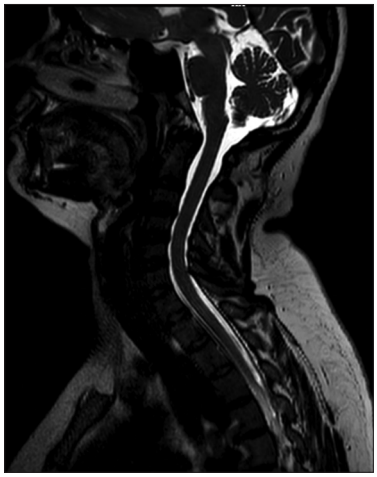

**Figure 3: Magnetic resonance imaging cervical spine taken after 3 months showed complete disappearance of the lesion with treatment**

Diagnosis is made by detection of the MOG antibody by cell-based serum assays [6]. Acute treatment is with pulse dose steroids followed by a taper of oral steroids. Disease-modifying drugs are used in relapsing cases and those with high antibody titers. These drugs include azathioprine, mycophenolate, and rituximab. Residual disability develops in up to 80% of patients, with transverse myelitis at onset being the most significant indicator of long-term prognosis [7].

In conclusion, MOG antibody disorder is an important cause of myelopathy in young patients and should be kept in the differential diagnosis. It can present with symptoms related to the spinal cord, brain, and optic nerves. Early diagnosis and treatment lead to good long-term outcomes.

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