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# Spinal Magnetic Resonance Imaging in a Patient with Systemic Lupus Erythematosus with Sensorimotor and Ataxic Neuropathy

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**Abstract** We present a patient with systemic lupus erythematosus and sensorimotor and ataxic neuropathy in whom magnetic resonance imaging revealed a high signal intensity in the posterior column of the cervical cord. MRI is useful in detecting the lesions of the posterior column due to a degeneration of the primary sensory neurons in patients with peripheral neuropathy.

*Key words*: Peripheral neuropathy, Sensory ataxia, Systemic lupus erythematosus, Magnetic resonance imaging, Posterior column

Patients with systemic lupus erythematosus (SLE) can develop nervous system involvement with a wide variety of clinical signs and symptoms. However, sensory ataxia due to peripheral neuropathy appears to be rare in such patients [1] . We present a patient with SLE with sensorimotor and ataxic neuropathy in whom MRI showed high signal intensity in the posterior column of the cervical cord.

## Case Report

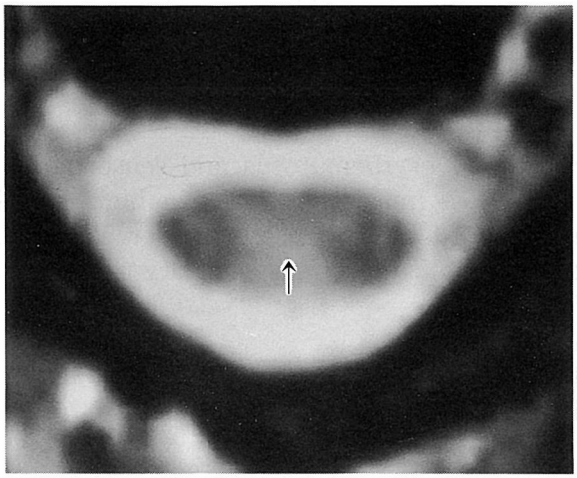
A 42-year-old Japanese woman had SLE, who had been diagnosed by the presence of high titers of anti-nuclear and anti-DNA antibodies, and by the presence of membranoproliferative glomeluronephritis. She developed muscular weakness of the extremities and a gait disturbance several days after suffering an upper respiratory infection, and was admitted to a local hospital with tetraparesis and paresthesias in the arms and legs. Two months later, her muscular weakness was improved, but the numbness and paresthesias in her arms and legs had gradually become worse. She was therefore transferred to our hospital.

Neurological examination on admission revealed mild muscular weakness of the upper and lower limbs on both sides, being worse on the left. She was able to walk only with assistance. There was a mild disturbance of touch and pain sensation mainly on the left leg and arm. Marked loss of position sense in the left arm was accompanied by pseudoathetosis. Position sense was less impaired in the left arm and both legs. Disturbance of vibration sense followed a similar pattern. Romberg's test was positive. She had sensory ataxia of the left arm and both legs. Deep tendon reflexes were absent in the left arm and both legs, and decreased in the right arm without Babinski's sign.

Laboratory studies demonstrated mild anemia, high titers of antinuclear antibody and

antibodies to single stranded DNA. Serum anti-Hu antibody, SS-A and SS-B antibodies were negative. General examination including chest X-ray, chest and abdominal CT, and gastrointestinal endoscopy revealed no malignancy. There was proteinuria, but renal function was normal. The cerebrospinal fluid was normal.

Nerve conduction studies demonstrated an absence of sensory nerve action potentials in the ulnar nerves bilaterally, the left median nerve, and the left sural nerve. Sensory conduction velocity of the right median nerve and the right sural nerve was mildly slowed to 53.4m/sec and 46m/sec, respectively. Motor nerve conduction velocity was slowed to 39.1m/sec in the left posterior tibial nerve and to 43.2m/sec in the left median nerve. Somatosensory evoked potentials (SEPs) were not elicited by stimulation of the left median nerve. SEPs elicited by stimulation of the right median nerve were normal. No biopsy of the sural nerve was obtained.



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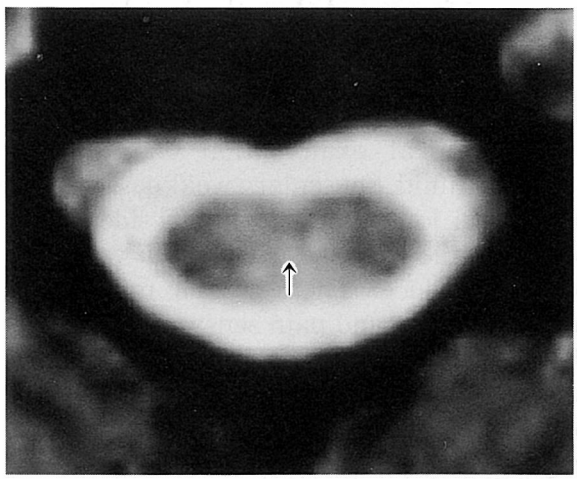

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Fig. 1 Axial T2*-weighted gradient echo -images (TR/TE 470/18, Flip angle15°) of cervical cord at the level of C5(a) and C7(b) vertebral body. A high intensity signal is present in the posterior column (←)

She was treated with intravenous methylprednisolone 1000mg daily for 3 days, and in two weeks the muscular weakness and sensory ataxia mildly improved. She became able to walk without assistance. However, her neurological symptoms persisted for the next 3 years of observation. Neurological examination 3 years after disease onset revealed slight muscular weakness of the left upper and lower limbs. There was a mild disturbance of touch and pain sensation mainly on the left leg and arm. Moderate loss of position sense in the left arm was accompanied by pseudoathetosis. Romberg's test was equivocal. Deep tendon reflexes were absent in the left arm and both legs, and decreased in the right arm without Babinski's sign.

MRI performed on 1.5 tesla unit demonstrated a high signal intensity in the posterior column of the cervical cord at the level of C4 -C7 vertebral bodies on T2*-weighted gradient echo-images [Fig. 1] . T1-weighted images showed no abnormality.

## Discussion

Patients with SLE can develop nervous system involvement. Peripheral neuropathy develops in 6% to 21% of patients with SLE [2-4] . These are usually distal symmetric polyneuropathies with predominantly sensory symptoms and subacute or chronic evolution [2,3,5]. Rarely is peripheral neuropathy an initial symptom in SLE. Although sensory ataxia due to peripheral neuropathy is rare, Sadeh et al reported a case of SLE with sensory ataxia as an initial symptom [1] . Since our patient developed sensorimotor neuropathy during SLE and had no other disorders that would cause neuropathy, we

believe it was due to the SLE.

Sensory ataxia due to neuropathy has been caused by ataxic or pseudotabetic Guillain-Barré syndrome, intoxication with drugs such as penicillin, cisplatin, and pyridoxine [6], carcinomatous sensory neuropathy [6, 7], acute autonomic and sensory neuropathy [8,9], Sjögren's syndrome [10], and chronic idiopathic ataxic neuropathy [6]. Patients with these diseases are considered to have a sensory neuronopathy involving the dorsal root ganglions [6-10]. Degeneration of the dorsal root ganglion and the posterior column in the spinal cord has been demonstrated at autopsy in some cases [7-9]. Yamamoto et al recently described a case of carcinomatous neuropathy with degeneration of the posterior column detected on MRI [11]. Yasuda et al reported a case of acute autonomic and sensory neuropathy in which MRI revealed a degeneration of the posterior column [12].

In our patient, the finding of SEPs, sensory ataxia, and a persistent sensory disturbance, especially prominent in the deep sense, suggested an involvement of the dorsal root ganglion neurons and the posterior column. High signal intensities in the posterior column may demonstrate retrograde degeneration, such as demyelination or/and axonal loss due to an involvement of the dorsal root ganglion neurons. We believe that high signal intensities in the posterior column of the cervical cord on T2*-weighted images imply an involvement of the central axons of the sensory ganglion neurons in such patients with sensory neuropathies.

MRI is useful in detecting lesions of the posterior column due to degeneration of the primary sensory neurons in a patient with peripheral neuropathy.

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