Teaching Neuro*Image*: The L5 spinal cord segment

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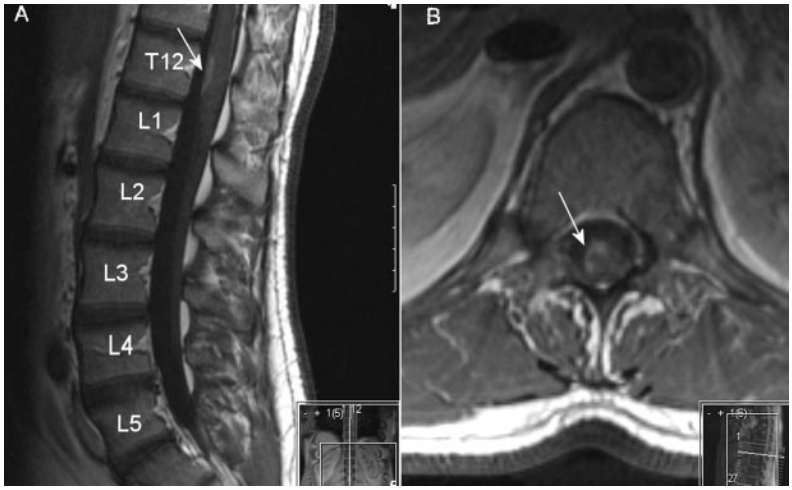
A 35-year-old man noted an abrupt painless right foot drop with severe weakness of ankle dorsiflexion, inversion, and eversion. Plantar flexion was normal. Deep tendon reflexes and sensation were normal. Straight leg raise was negative. Electrodiagnostic studies, performed 2 days after onset of weakness, revealed acute severe L5 radiculopathy, as evidenced by a markedly decreased recruitment (i.e., decreased number of motor unit action potentials firing rapidly) in all L5 innervated muscles with normal unit configuration and no fibrillation potentials. Spinal MRI revealed an enhancing right hemicord lesion at the level of T12 vertebra (figure). Cerebrospinal fluid examination was normal. The foot drop resolved and the cord lesion disappeared after a 3-day course of IV methylprednisone (1,000 mg/day). A year later, the patient developed numbness in the left leg and left hemitrunk and had a left Babinski sign. MRI of the brain revealed several new periventricular white matter lesions, consistent with multiple sclerosis. The patient was started on interferon beta-1a and has been stable for 2 years.

Foot drop is defined as severe weakness of ankle dorsiflexion with intact plantar flexion. Common causes of unilateral foot drop include peroneal neuropathy, L5 radiculopathy, sciatic nerve lesion, or lumbosacral plexopathy.[1] Foot drop due to L5 myotomal involvement is rarely caused by a focal segmental cord lesion. There are three previously reported patients with MS presenting as foot drop due to L5 motor radiculopathy.[2-4]

The clinical and electrodiagnostic findings in this patient pointed to a lower motor neuron lesion affecting the L5 root motor axons, and the imaging studies showed that this was due to a segmental cord lesion involving the root exit zone. In this patient as well as one previous patient,[4] there was anatomic–radiologic correlation, and the plaque involved the lateral section of the spinal cord, likely including the emerging L5 motor fibers. The location of the L5 cord segment in both patients was across the T12 vertebra.

The discrepancy between the vertebral and spinal cord levels increases further down the spinal column.[5] The vertebral column elongates more rapidly than the spinal cord during fetal development and childhood. Because the cord is fixed rostrally, the caudal end opposes to the L2–3 disc at birth. In adults, the spinal cord ends at the L1–2 disc space level, but this may vary from the body of T12 to the body of L3.

Figure T1-weighted MRI of the lumbar spine after gadolinium enhancement



T1-weighted MRI of the lumbar spine after gadolinium enhancement, revealing a enhancing segmental cord lesion across T12 vertebral body (arrows) which involves the white matter and the root exit zone on the right. This corresponds to the exact location of the L5 cord segment in this patient. Sagittal (A) and axial views (B).

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