Rapidly-progressive Paraparesis Secondary to HTLV-I Associated Myelopathy/Tropical Spastic Paraparesis: A Case Report

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Case Description

49-year-old Jamaican woman with no history presents with rapidly-progressive spastic paraparesis of bilateral lower extremities (BLE). Her symptoms started with ascending pedal pins-and-needles sensation, progressing to BLE weakness, stiffness and spasms within 2 months, and loss of sensation and active BLE motor function with bowel and bladder incontinence within 3 months. Medical care was delayed due to COVID-19 pandemic, resulting in BLE deep vein thrombosis.

Physical exam significant for spasticity without volitional movement of BLE, right foot non-sustained clonus, left great-toe proprioception deficit, decreased pain/temperature/vibratory sensation below right T8 and left T10 levels, and BLE hyperreflexia. MRI spine demonstrated T2-weighted heterogenous cord signal abnormality along T5-T9 levels. Complete neurologic and rheumatologic workup negative, except for positive methylmalonic acid, cardiolipin antibodies, and HTLV-I/II serum antibodies.

Discussion

HTLV-I virus is endemic in the Caribbean, Japan, and Africa, affecting people between 30-50 years. Transmission occurs by breastfeeding, sexual intercourse, blood transfusion, and intravenous drug use. HAM/TSP is rare, affecting about 2% of carriers. A suggested mechanism of acute symptom progression points to an inflammatory process linked to the cell-mediated immune process in the spinal cord. Progression occurs slowly with disease onset to wheelchair confinement occurring within a median of 21 years.

Clinical features include progressive lower extremities weakness and spasticity, hyperreflexia, low back pain, detrusor instability, and sensory changes. Diagnostic testing includes imaging, CSF and serum testing, and PCR. MRI of the brain and spinal cord may be normal or show cervical/thoracic spinal cord atrophy. Current treatment is limited to management of symptoms. Disease-modifying agents with promising results include corticosteroids, danazol, pentoxifylline, interferon-beta-1a, plasmapheresis, and anti-CCR4 monoclonal antibody.

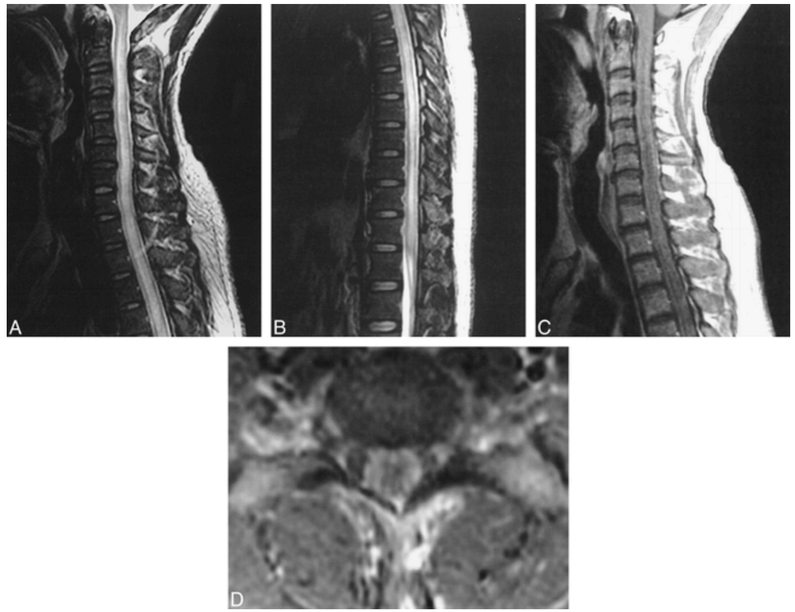

Figure 1. Sagittal T2-weighted images reveal marked cord swelling and high intensity in the central portion over the entire length of the spinal cord (A and B). Peripheral enhancement is shown by a sagittal T1-weighted image (C) and an axial T1-weighted image at the C7/T1 level (D) with contrast material

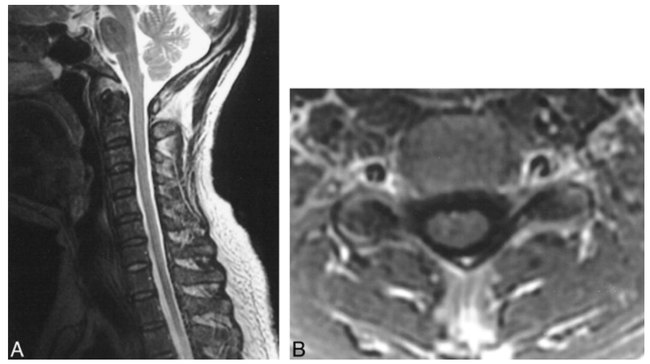

Figure 2. Three years after admission, the spinal cord has no signal abnormality on the T2-weighted image (A). Mild cord atrophy is visible on the contrast-enhanced, axial T1-weighted image at the C5/6 level (B)

Conclusion

Although electrodiagnostic testing was negative for LEMS and malignancy workup unremarkable, the patient's history of smoking would be reason to consider a paraneoplastic syndrome related to small cell lung cancer. It is, however, possible that her condition is not related to paraneoplastic disease as calcium channel antibodies may be positive in both paraneoplastic and non-paraneoplastic cerebellitis.

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*Images do not reflect the condition of the patient and are for illustration purposes only.