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Human T-cell lymphotropic virus, type 1 (HTLV-1)-associated myelopathy: MR findings

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We present a case of human T-cell lymphotropic virus, type 1 (HTLV-1)-associated myelopathy, also known as tropical spastic paresis. While the literature has previously described the neuroradiological findings of spinal atrophy and hyperintense T2 lesions involving the cerebral white matter, little information is available about musculoskeletal imaging findings. Our patient demonstrated diffuse muscle atrophy of the thigh muscles, particularly the posteromedial groups. Our case supports HTLV-1-associated myelopathy as a disease of neurological etiology with resulting muscular denervation.

Case report

A 46-year-old man with human T-cell lymphotropic virus, type 1 (HTLV-1), diagnosed by serum and CSF analysis, presented with a 7-year history of slowly progressive, bilateral, lower-extremity weakness and tightness. His stiffness predominantly involved the lower calves and, to a lesser extent, the upper thighs. These symptoms were causing difficulty ambulating. Despite several years of treatment with antiviral therapy and steroids, the patient's condition progressively worsened. On neurological examination, there was reduced strength in his thighs and calves, inturned toe-walking with cane assistance, and decreased pinprick sensation in a stocking-glove distribution to the knees and mid-forearms bilaterally. The patient also had several years of urinary dysfunction, with bladder urgency and spasticity. MRI of the spine from an outside hospital was reported as normal.

MRI of the left thigh with gadolinium was obtained to further evaluate his symptoms. Diffuse fatty atrophy was present in the adductor magnus, semimembranosus, semitendinosus, and vastus lateralis muscles of the left thigh on T1-weighted images (Figs. 1 and 2). No abnormal signal or

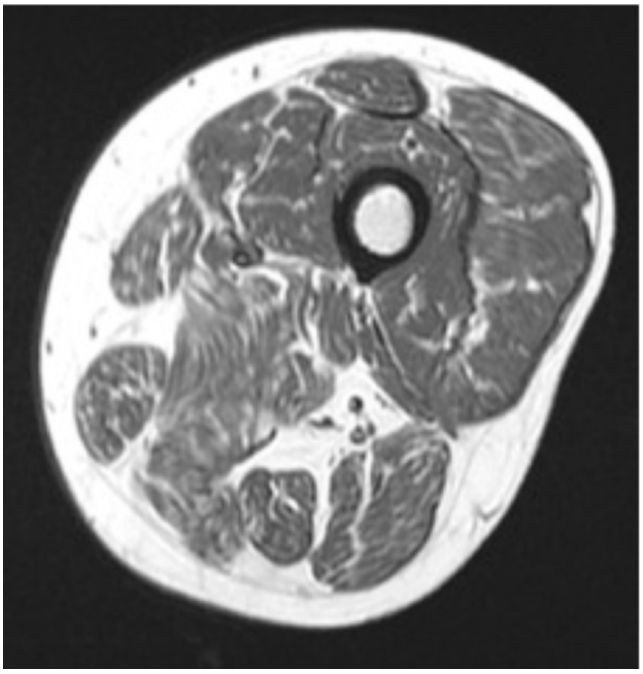

Figure 1. 46-year-old male with HTLV-1-associated myelopathy. Axial T1-weighted MR of the left thigh shows fatty atrophy involving the adductor magnus, semimembranosus, and semitendinosus muscles.

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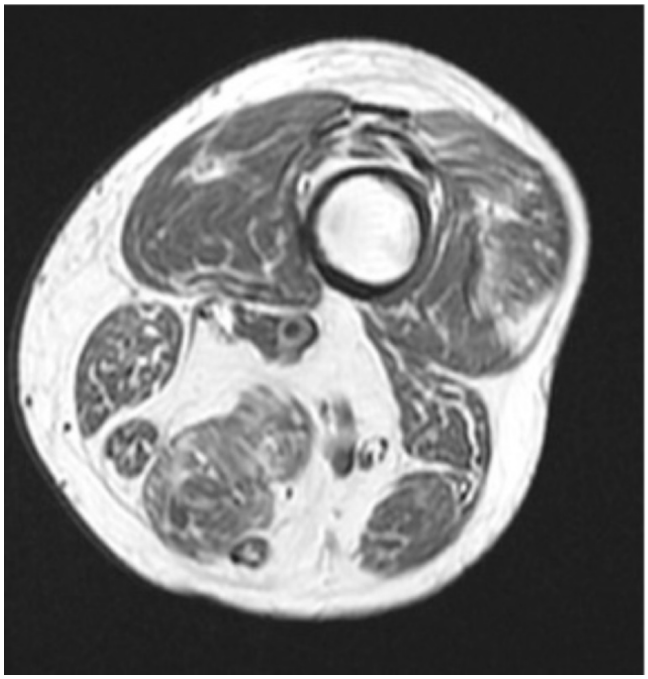

Figure 2. 46-year-old male with HTLV-1-associated myelopathy. Axial T1-weighted MR of the more distal left thigh shows fatty atrophy involving the semimembranosus, semitendinosus, and vastus lateralis muscles. There is partial atrophy of the vastus lateralis muscle.

enhancement was noted on short tau inversion recovery (STIR) images (Fig. 3). Electromyogram (EMG) and muscle biopsies were not obtained. The underlying etiology of the MR finding of fatty atrophy was diagnosed as HTLV-1 myelopathy/tropical spastic paresis due to combination of clinical findings of spastic paraparesis, urinary dysfunction, and positive HTLV-1 antibodies.

Discussion

HTLV-1 is a retrovirus endemic to Japan and the Caribbean (1). HTLV-1 infects the body's CD4 cells, while HTLV-2 infects CD8 cells (2). There are several routes of viral transmission, including sexual, parenteral, transfusion, contamination of needles, and breastfeeding (3-6). The majority of patients remain asymptomatic. However, the disease is associated with adult T-cell leukemia/lymphoma (ATLL), which is an aggressive malignancy with low (6-month) survival rates (7). Several other diseases are associated with HTVL-1 infection, including pneumopathy, uveitis, eczema, xerosis, folliculitis, and myelopathy (2, 8-10).

HTVL-1 associated myelopathy (HAM) is also known as tropical spastic paraparesis (TSP) (11). Its prevalence is approximately 0.25% (12). Patients with HAM/TSP present with spastic paraparesis (particularly in the lower extremities), mild disturbances of sensation, and urinary dysfunction (13, 14). The muscle weakness and atrophy usually involve the proximal muscle groups. The disease is chronic, with no known successful treatment. Possible etiologies for HTVL-1-associated myelopathy include leukemic cell inflammation of nerves, invasion of peripheral nerves, or autoimmune mechanism (15).

Previous cases demonstrating the imaging findings of HTLV-1 myelopathy have reported abnormal enlargement and gadolinium enhancement in the peripheral nerves (15). Increased 18-fluorodeoxyglucose (FDG) positron-emission tomography (PET) uptake has also been reported (15). The other case of musculoskeletal findings in HAM/TSP demonstrated atrophy within the semimembranosus, semitendinosus, adductor magnus, biceps femoris, and vastus intermedius and lateralis muscles (16).

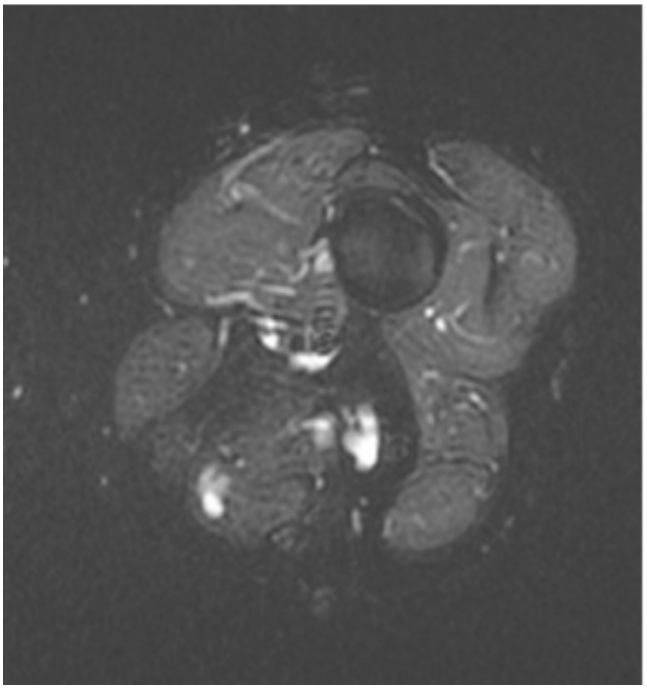

Figure 3. 46-year-old male with HTLV-1-associated myelopathy. Axial short tau inversion recovery (STIR) image of the mid left thigh demonstrates no abnormal signal in the musculature.

Similarly, our patient's pattern of atrophy involved the posteromedial and posterolateral muscles of the thigh. In the spine, reported MRI findings have included atrophic changes in the spinal cord (17). Additionally, multifocal high-signal-intensity lesions have been noted in the cerebral white matter on T2-weighted images (18). Thus, the muscle atrophy appears to have both central and peripheral neurological etiologies. While our case does not include EMG or biopsy results to confirm the diagnosis, the clinical features of spastic paraparesis and urinary dysfunction are most consistent with HAM/TSP. Ultimately, our case provides another differential diagnostic consideration for patients with muscle denervation on MRI.

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