Lhermitte’s Sign in Alcoholic Myelopathy without Portosystemic Shunting: MRI Evaluation

Abstract

We conducted spinal MR imaging on a 35-year-old man with Lhermitte’s sign that had manifested over the previous 4 years. He had consumed more than 500 ml of whisky daily for at least 10 years. However, he did not show any evidence of severe liver disease with hepato-systemic blood shunting. Neurologic examination revealed markedly depressed sense of vibration in the feet and mild spasticity in the lower limbs, together with Lhermitte’s sign. MR imaging revealed abnormal signal intensity in the posterior column spanning the whole length of the upper cervical cord, which is consistent with Lhermitte’s sign.

Key words: Lhermitte’s sign, posterior column, cervical cord, alcoholic myelopathy, MRI

Introduction

Myelopathy associated with alcoholism usually occurs in patients who heavily consume alcohol and have cirrhosis and portosystemic blood shunting (1, 2). On the other hand, Sage et al (3) proposed another syndrome of progressive posterior and lateral column dysfunction in alcoholic myelopathy without substantial liver disease. The myelopathy usually begins with sensory disturbance of the lower limbs followed by progressive spastic paraparesis with clinical signs of both lateral and posterior column involvement. We describe a patient with alcoholic myelopathy presenting Lhermitte’s sign (4) as a unique clinical sign consistent with the MRI finding of posterior column degeneration.

Case Report

A 35-year-old male dentist was referred to our hospital because of sudden electric-like sensations on flexion of the neck, which spread down the body, to the back, or to the extremities, so-called “Lhermitte’s sign”. He had had this condition for the previous 4 years. He had been consuming more than 500 ml of whisky daily for at least 10 years prior to onset of the condition. There was no family history of atopic diathesis. On general physical examination, he was fully conscious, well-nourished, and showed no sign of hepatic disease. Neurologic examination revealed a markedly depressed sense of vibration in the feet and spasticity in the lower limbs. Hyperactive deep tendon reflexes were observed in the legs without Babinski’s signs. Position sense was affected but minimally in the toes, and he showed no ataxic gait. Muscle strength and superficial sensation in the extremities were not affected. Cranial nerves and cerebellum were intact. Laboratory evaluation revealed elevated AST (88 IU/l) and ALT (47 IU/l), but otherwise normal liver function parameters. Serologic tests for syphilis were negative. Serum vitamin B12 (930 pg/ml) and folate (2.9 ng/ml) levels were within normal limits.

Electrophysiologic examination revealed markedly prolonged central conduction time of somatosensory evoked potentials (SEPs) when the tibial nerve was stimulated. Peripheral nerve conduction studies and SEPs by median nerve stimulation were essentially normal. Sensory nerve conduction velocities calculated on the right side were as follows: 50 m/s for median nerve, 51 m/s for ulnar nerve, and 49 m/s for sural nerve.

Computed tomography showed round fatty infiltration on both lobes of the liver. Dynamic computed tomography scan showed neither tumor shadow nor abnormal systemic collateral. Ultrasonography demonstrated a fatty liver without reversal of portal vein flow, ascites or intra-abdominal collaterals.

MRI T2 studies revealed abnormal high intensity signal in the posterior column spanning the whole length of the upper cervical cord (Fig. 1). Cord swelling was not evident in successive transverse sections of cervical MRI. No abnormal signal was detected in any other tract of the cervical cord or in any other part of the central nervous system.

Lhermitte’s sign disappeared within six months with abstinence from alcohol. Lumbar puncture was not possible during the entire course of illness because of refusal of the procedure by the patient.

Discussion

The present patient showed Lhermitte’s sign rather than paresthesia of the feet, which is typically recognized as an initial symptom of the myelopathic syndrome proposed by Sage et al (3). Nevertheless these initial clinical phenomena result from posterior column degeneration, which is sometimes observed in the spinal cord of alcoholics at necropsy (5). Although Sage et al (3) did not indicate any specific lesions in the spinal cord; our MRI findings clearly demonstrated an upper cervical lesion of myelopathy. It is controversial to explain the discrepancy between the upper cervical lesion and clinical features sparing the upper limb involvement. Our SEP studies also indicated that the ascending sensory conduction was normal at the median nerve, but was markedly affected at the tibial nerve. One possible explanation for the discrepancy is that the affected axons are more
severely damaged in the more distal sites (dying-back process) as proposed for posterior column degeneration (6), in which the centrally-directed axons of the posterior column from the lumbar cord are more severely affected than those from the cervical cord. Although there is no conclusive evidence for the dying-back process of posterior column degeneration in the present case, the predominant pathological process of alcoholic peripheral neuropathy is considered to be an example of dying-back neuropathy (5). Unfortunately, there are no pathological studies of cases purported to be alcoholic myelopathy because this condition could be masked by other common neurological complications of alcoholism (1, 5). Our report indicates that MRI is useful for the diagnosis of spinal cord involvement in alcoholics.

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