Teaching Video NeuroImages: Hepatic myelopathy

An unusual neurologic complication of hepatic encephalopathy

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Figure Imaging and spectroscopy



(A) T1-weighted axial brain MRI shows hyperintense signal in internal globus pallidus (manganese deposition due to hepatic encephalopathy [HE]).(B) MRI spectroscopy with water-suppressed proton spectra of a voxel located in normal-appearing parietal white matter shows an increase in glutamine/glutamate signal intensity (Glx, 2.15–2.45 ppm) associated with a decrease in myo-inositol (Ins, 3.45 ppm) and choline (Cho, 3.2 ppm). N-acetylaspartate (NAA) index has normal signal intensity (2.0 ppm). These findings are typically observed in HE.

A 57-year-old man with alcoholic cirrhosis presented with a 4-year history of hepatic encephalopathy (HE) and progressive gait impairment that worsened during HE (video 1). Examination disclosed spasticity and weakness in lower limbs. Brain MRI showed hyperintense signal in basal ganglia and spectroscopy disclosed increased glutamine/glutamate and decreased myo-inositol (figure). Spine MRI was normal, and other causes were ruled out. Hepatic myelopathy was diagnosed.

Hepatic myelopathy is an unusual complication of liver disease and is characterized by progressive spasticity and lower limbs weakness over the years.¹ Symptoms usually worsen during HE episodes. Treatment of HE and liver transplantation may partially improve gait.²

Author contributions

V.B. Ciarlariello: case report project: conception, organization, execution; manuscript: writing of the first draft, review and critique. M.V.T. Fujino: case report project: conception, organization, execution; manuscript: writing of the first draft, review and critique. M.D.d. Almeida: case report project: conception, organization, execution; manuscript: review and critique. O.G.P. Barsottini: case report project: conception, organization, execution; manuscript: review and critique. J.L. Pedroso: case report project: conception, organization, execution; manuscript: review and critique.

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