Acquired pendular nystagmus from cerebellar nodulus acute ischemic lesion

Figure MRI

Fast spin-echo T2-weighted axial (A) and fluid-attenuated inversion recovery (B) MRI show a slight hyperintensity signal (asterisk) in the nodular portion of the cerebellar vermis. Diffusion-weighted imaging reveals a signal hyperintensity of the affected area (C), with a decrease in apparent diffusion coefficient, indicating cytotoxic edema (D).

A 71-year-old woman developed acute ataxia and oscillopsia, with underlying horizontal, binocular, conjugate pendular nystagmus (PN), with an amplitude of 4° in both eyes and a 3 Hz frequency, without gaze-evoked components.

Brain MRI detected an altered signal in the nodular portion of cerebellar vermis, indicating cytotoxic edema (figure), due to an ischemic lesion.

Acquired PN has been traditionally attributed to “Guillain-Mollaret triangle” lesions. Recently, a neural integrator has been hypothesized in the brainstem, which guarantee the ability to hold steady eccentric gaze, whose disruption may give rise to PN.¹

This case supports the contribution of vestibulocerebellum, mainly the nodulus, to modulate such neural network.²

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