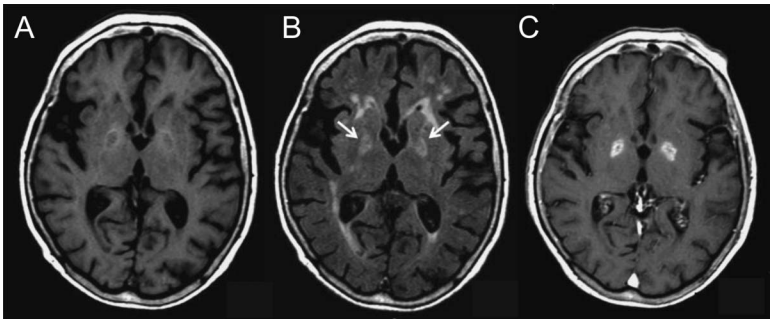
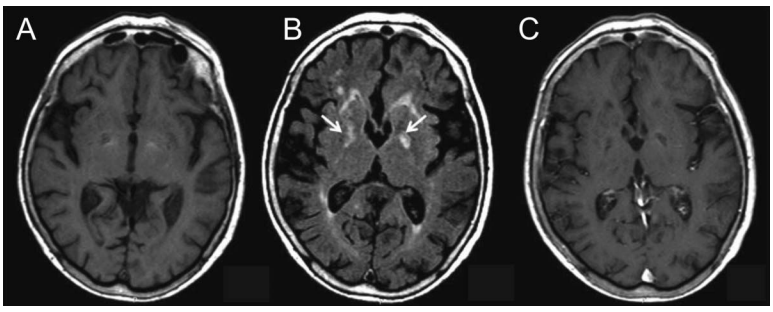
Isolated bipallidal lesions caused by extrapontine myelinolysis

Figure 1 Brain MRI performed 12 days after the onset of symptoms



Brain MRI shows bipallidal lesions, hypointense in T1-weighted images (A), hyperintense (arrows) in T2–fluid-attenuated inversion recovery (B), with contrast enhancement after gadolinium administration (C). Evidence of chronic small-vessel ischemic disease was also present (B).

Figure 2 Brain MRI after 2 months



Follow-up MRI scans reveal bipallidal hypointensity on T1-weighted images (A), bipallidal hyperintensity (arrows) on fluid-attenuated inversion recovery images (B), and the absence of contrast enhancement (C). A follow-up neurologic examination 2 months after the initial presentation reveals significant clinical improvement.

An 89-year-old woman developed bradykinesia, mutism, and apathy after a rapid correction of hyponatremia caused by repeated vomiting. Brain MRI showed bipallidal involvement that improved at follow-up (figure 1, figure 2). The patient's clinical history and neuroimaging are suggestive of extrapontine myelinolysis. This disease involves basal ganglia but the globus pallidus is usually spared or not singly involved.[1] A patient with bipallidal extrapontine myelinolysis has been previously described.[2] Toxic, hypoxic, and metabolic causes of bipallidal involvement were excluded in our patient. Extrapontine myelinolysis should be included in the differential diagnosis of patients with a history of hyponatremia, subacute parkinsonism, and bipallidal lesions on MRI.

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