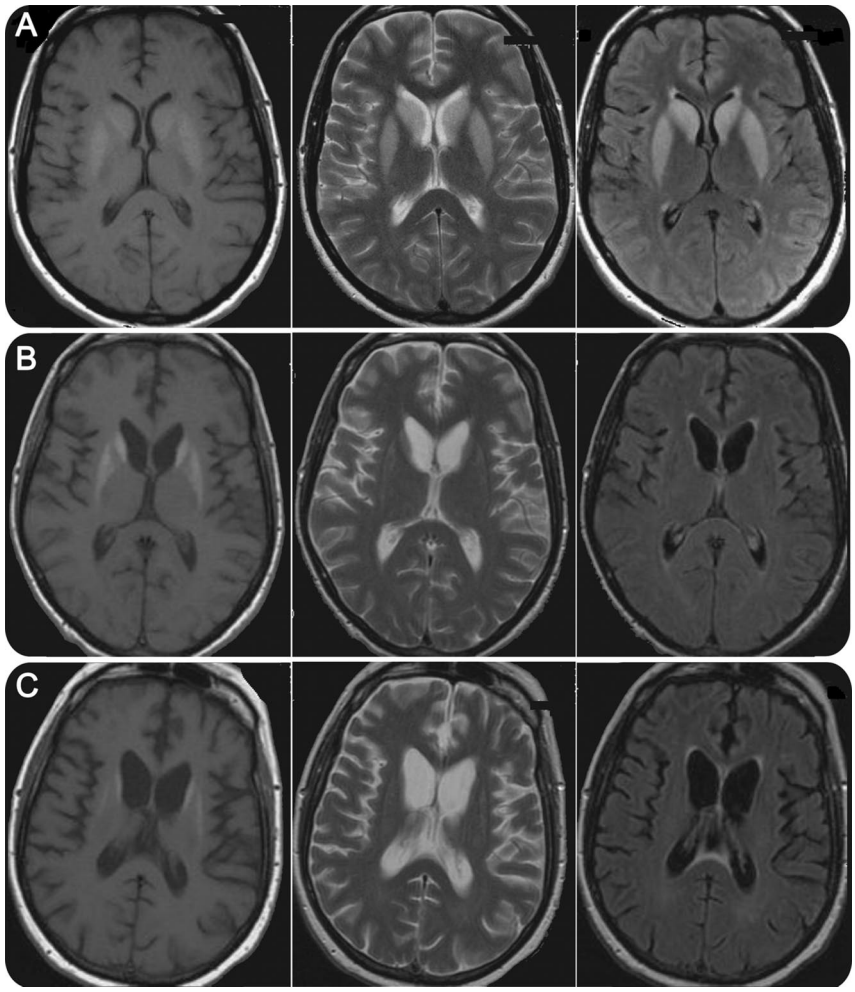
Postencephalitic parkinsonism and basal ganglia necrosis due to Epstein-Barr virus infection

Figure Brain MRI



Axial brain MRI showing T1-weighted (left column), T2-weighted (middle column), and fluid-attenuated inversion recovery (right column) sequences at 1 week (A), 6 months (B), and 12 months (C) after symptom onset. Note selective hyperintensities of the caudate nuclei, putamen, and external portion of the globus pallidum bilaterally in both T1- and T2-weighted sequences, with progressive atrophy of these deep nuclei over time. Ventricular enlargement results from striatal volume loss. There was no diffusion restriction at any timepoint (not shown).

A 35-year-old woman with hypertension, pancreatitis, and idiopathic renal disease developed decreased verbal output, sleepiness, and disorientation shortly after renal transplantation. MRI demonstrated striatal hyperintensity (figure, A). CSF showed 32 leukocytes (100% lymphocytes) and normal protein (47 mg/dL) and glucose (68 mg/dL). Tacrolimus levels were not elevated. Epstein-Barr virus (EBV) DNA was detected by PCR (1.5×10^2 copies/mL). She evolved to a stable state of akinetic-rigid mutism with apraxia of eyelid opening (video on the *Neurology*® Web site at www.neurology.org). MRIs at 6 and 12 months (figure, B and C) showed progressive putaminal and caudate atrophy. Levodopa and ropinirole were ineffective. EBV causes postencephalitic parkinsonism,[1] possibly due to cross-reactivity of EBV antibodies with α-synuclein, a pathogenic Parkinson disease protein.[2]

Supplemental data at www.neurology.org

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