A 50-year-old man presented with a 3-day history of confusion. He had a previous medical history of alcohol abuse and arterial hypertension. He received therapy with combined hydrochlorothiazide and ramipril. On admission, the patient was not febrile (37.1 °C); physical examination was otherwise normal. Laboratory studies disclosed: serum sodium: 109 mEq/l, erythrocyte sedimentation rate: 6 mm/h, C-reactive protein: 3 mg/l, white blood cell count: 5.4 g/l. Other routine biochemical tests, including kaliemia, blood glucose level, renal and liver tests, erythrocyte sedimentation rate, C-reactive protein, blood protein electrophoresis, were normal. Brain computed tomography scan was normal. The patient was administered intravenous infusion of normal saline solution, which resulted in improvement of confusion and normalization of the serum sodium level within 2 days after his admission. Two weeks later, the patient was readmitted for recurrence of confusion; he was not febrile (37.2 °C). Physical examination also revealed dysarthria, lumb and trunk ataxia and brisk deep tendon reflexes. Cerebrospinal fluid analysis showed: white cell and red cell counts <2/mm³, protein level: 0.25 g/l, glucose level: 3.4 mmol/l, chloride level: 120 mmol/l; Gram staining and bacterial and fungal cultures of the cerebrospinal fluid were negative. Brain magnetic resonance imaging (MRI) was performed, showing changes consistent with central pontine myelinolysis (Figures 1a and b).

First described by Adams in 1959, central pontine myelinolysis is an uncommon non-inflammatory disease of the white matter tracts traversing the pons.² The condition has commonly been reported in patients with chronic alcoholism, cirrhosis, malnutrition and severe burns, who experienced rapid correction of slowly progressive hyponatremia.³ Our case confirms that brain MRI is a non-invasive test that is helpful for providing both detailed and complete cartography of brain changes in patients with central pontine myelinolysis. In this instance, brain MRI showed: (i) characteristic ‘owl’s eye’ pattern on axial T2-weighted images; and (ii) typical abnormalities...
involving the basal pons resembling the face of a monkey, defining the ‘monkey sign’.3–5

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References