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Temporal Changes of MR Findings in Central Pontine Myelinolysis

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Summary: We report central pontine myelinolysis in orthotopic liver transplant patients. Sequential MR imaging of these patients with central pontine myelinolysis shows progressive decrease of T2-weighted MR signal in the pons, which may not resolve despite complete neurologic recovery.

Index terms: Myelinolysis; Brain, magnetic resonance; Pons; Demyelinating disease

Central pontine myelinolysis is a demyelinating disorder that almost exclusively affects the central portion of the basis pontis. Central pontine myelinolysis was described by Adams et al (1) in 1959 in patients with a history of alcoholism and malnutrition. The cause of central pontine myelinolysis remains unclear, but many studies (2–8) have implicated changes in serum sodium, specifically the rapid correction of hyponatremia or overcorrection to hypernatremia. A subgroup of patients at risk for central pontine myelinolysis are those undergoing orthotopic (grafted into its normal position) liver transplantation. Transplantation encephalopathy (2) and/or rapid serum sodium changes may be responsible for the 10% to 13% incidence of central pontine myelinolysis in orthotopic liver transplant patients (2, 3, 5).

We present three cases of central pontine myelinolysis in orthotopic liver transplant patients.

Subjects and Methods

From December 1984 through February 1991, 20 patients underwent orthotopic liver transplantation at our institution. As part of an evolving pretransplant imaging protocol, the most recent 8 of the 20 patients had pretransplantation brain magnetic resonance (MR) studies. Serial posttransplantation MR studies were performed in both healthy and neurologically abnormal patients.

In 5 of the 20 patients, neurologic symptoms developed after surgery. Two patients, had MR and clinical evidence

of stroke. Neither of these 2 patients had a pontine abnormality demonstrated on T2-weighted images. In the other 3 patients (3 women, 37 to 58 years of age), central pontine myelinolysis was diagnosed clinically. Each of the 3 patients had undergone rapid perioperative correction of hyponatremia. All 3 patients did show abnormal MR findings in the pontine portion of the brain stem (Figs 1 and 2). Abnormalities were not seen on T1-weighted images. In all 3 patients with central pontine myelinolysis, there was, over time, complete resolution of symptoms in the face of persistent but decreased brain stem lesion size.

Discussion

Initial signs and symptoms of central pontine myelinolysis typically include mental status changes, seizures, bradypnea, spastic paresis, quadriparesis, and pseudobulbar palsies. The rapid development of a “locked-in” syndrome also can occur in patients with large lesions in the basis pontis (2–6). Tracts that may exhibit demyelination include the pyramidal tracts (corticospinal and corticobulbar), which can lead to hyperreflexia, Babinski’s sign, quadriparesis, and, ultimately, quadriplegia. The corticopontine and pontocerebellar fibers also may be involved.

Among the several factors proposed as a cause of central pontine myelinolysis, the rapid correction of hyponatremia or overcorrection to hypernatremia most often is implicated. Major electrolyte disturbances often complicate orthotopic liver transplantation such that these patients are at risk for central pontine myelinolysis. The incidence of central pontine myelinolysis at our institution (15%) is similar to that of previous reports in patients undergoing orthotopic liver transplantation (2–4). All three of our patients with central pontine myelinolysis underwent rapid correction of their hyponatremia perioperatively (Figs 1 and 2).

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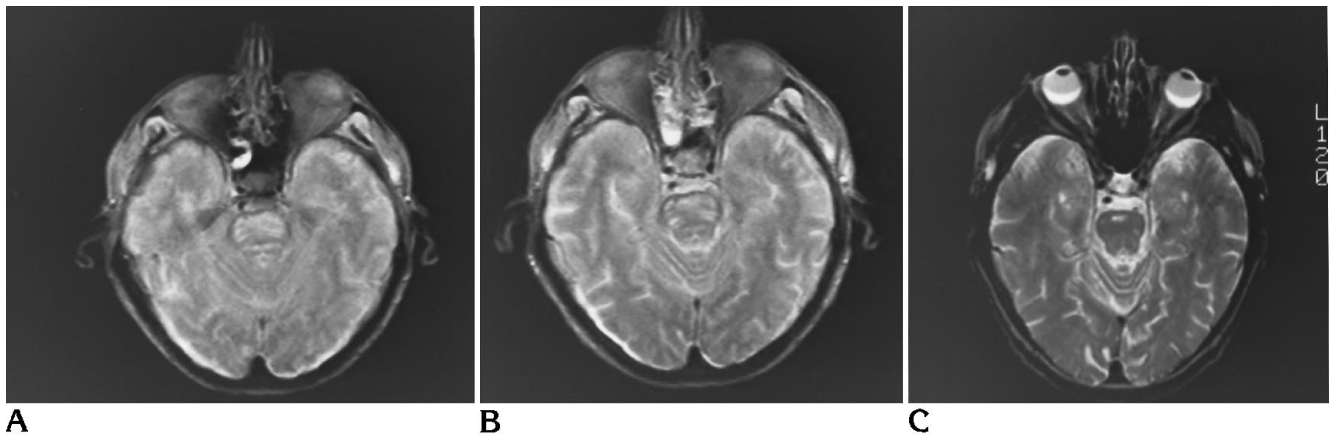


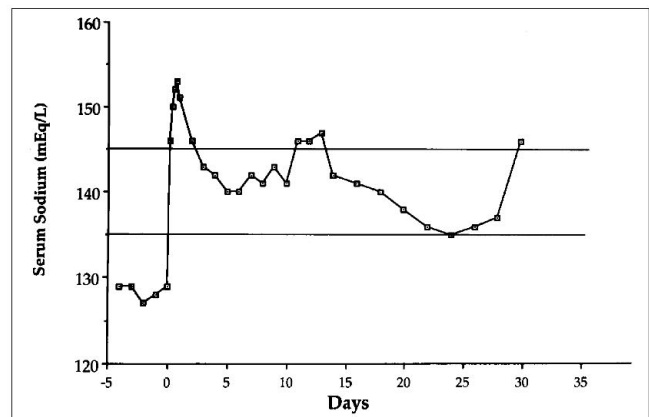
Fig 1. A 58-year-old woman underwent orthotopic liver transplantation for end-stage liver disease caused by sclerosing cholangitis. Before surgery, the patient was alert and without focal neurologic deficits. On the 4th postoperative day, aphasia developed and the patient was increasingly lethargic. Cranial computed tomography revealed a left frontoparietal subdural hematoma, but no pontine lesions.

A, Eight-day follow-up T2-weighted image of the brain shows diffuse abnormalities of the pons. The pontine lesions were unidentifiable on T1-weighted images. Repeat head computed tomography during this period with and without contrast material showed only a decrease in the size of the subdural hematomas. The patient's neurologic condition had slightly improved by the 4th week after orthotopic liver transplantation.

B, Follow-up axial T2-weighted image 4 weeks after orthotopic liver transplantation shows a decrease in area of involvement of the pons as compared with A. The patient continued to make progress during her hospital course with marked decrease in seizure activity.

C, At the 116th week after transplantation, axial T2-weighted image shows further reduction in area of involvement but persistent focal signal abnormalities.

D, A marked overcorrection of this patient's hyponatremia occurred perioperatively. Daily postoperative serum sodium concentrations then fluctuated within the normal and high range.



In our patients with central pontine myelinolysis, we were unable to identify abnormalities in the basis pontis on T1-weighted MR images or on head computed tomography examinations. T2-weighted images were more sensitive in the detection of central pontine myelinolysis in each case. Each patient at the time of acute neurologic symptoms had moderate to marked increased T2-weighted signal in the pons (Figs 1 and 2). We believe T2-weighted MR sequences are more sensitive at identifying early and/or subtle pontine lesions. T2-weighted images can show early and/or mild central pontine myelinolysis lesions, as in our patients, which may be associated with a more favorable outcome. Unlike in previous studies, extrapontine myelinolysis and pontine atrophy were not noted in our patients (3, 6).

Our study has shown the temporal development and resolution of central pontine myelinolysis with MR imaging in three patients, which correlate with the clinical course of neurologic symptoms (Figs 1 and 2). Edema and/or demyelination probably are the cause of this appearance (9, 10). In the subacute or chronic phase of central pontine myelinolysis, these pontine lesions persist in a diminished form on T2-weighted images, and the patients may or may not have neurologic symptoms. Improvement in the appearance of these lesions may represent resolution of acute edema, remyelination, or decreased astrocytic response. We were unable to correlate the size of the pontine lesions on MR imaging with the specific degree of neurologic deficit; however, we found that as our patients' neurologic symptoms improved, the pontine le-

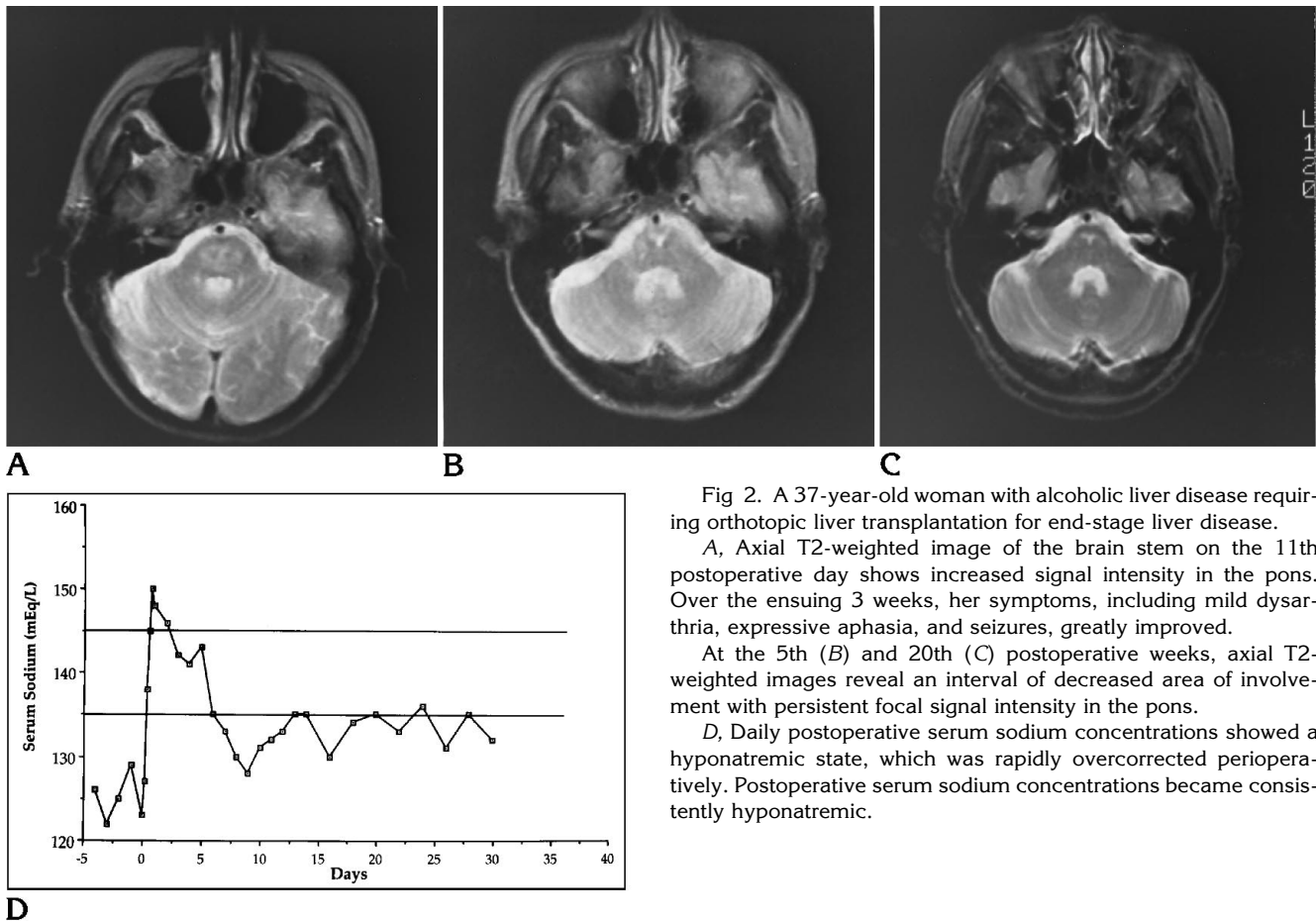


Fig 2. A 37-year-old woman with alcoholic liver disease requiring orthotopic liver transplantation for end-stage liver disease.

A, Axial T2-weighted image of the brain stem on the 11th postoperative day shows increased signal intensity in the pons. Over the ensuing 3 weeks, her symptoms, including mild dysarthria, expressive aphasia, and seizures, greatly improved.

At the 5th (B) and 20th (C) postoperative weeks, axial T2-weighted images reveal an interval of decreased area of involvement with persistent focal signal intensity in the pons.

D, Daily postoperative serum sodium concentrations showed a hyponatremic state, which was rapidly overcorrected perioperatively. Postoperative serum sodium concentrations became consistently hyponatremic.

sions decreased in size. These residual abnormalities represent areas of permanent damage.

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