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Subcortical White Matter Lesions in Osmotic Demyelination Syndrome

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Summary: We describe a case of histologically proved osmotic demyelination syndrome. In addition to abnormal T2 signal within the pons and thalami, MR showed linear enhancing lesions at the cortical medullary junction.

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

The osmotic demyelination syndrome is characterized by foci of myelinolysis that involve the pons (central pontine myelinolysis) but can also occur at other locations (extrapontine myelinolysis) such as the subcortical white matter.

Case Report

A 31-year-old alcoholic woman was admitted to our institution for seizures and confusion. On admission, her serum sodium was 105 mEq/L, and it was raised to 138 mEq/L in 12 hours with 3% saline. Her mental status improved promptly with disappearance of the seizures, and she was discharged after 5 days. Six days after discharge, she became confused again and progressively unresponsive for 12 hours. On readmission, she was drowsy and mute, opening her eyes when spoken to but not following commands. During the next week, progressive spastic quadriplegia developed. Thereafter, her neurologic condition did not change, and 7 weeks later she died of pneumonia.

Unenhanced computed tomography of the brain at the time of readmission showed a focal area of hypodensity in the pons. Magnetic resonance (MR) of the brain the next day showed an area of increase in signal intensity on T2-weighted images in the central portion of the pons with sparing of the periphery (Fig 1A). Hyperintense lesions were also present at the anterolateral portions of both thalami. T1-weighted images after intravenous administration of 12 ml of gadopentetate dimeglumine (0.1 mmol/

kg) showed ring enhancement of the pontine lesion and revealed multiple small ovoid or streaky enhancing lesions in the subcortical white matter of the frontal, temporal, parietal, and occipital lobes (Fig 1B). Some of these lesions were barely visible as hyperintense foci on fast spin-echo proton density- and T2-weighted images (Fig 1C).

At autopsy, marked softening of the pons was noted. The microscopic examination showed demyelination and lipid-laden macrophages in the central pons with preservation of neurons and axons. Similar lesions were identified in the subcortical white matter of the temporal, parietal, and occipital lobes and in the frontal opercula. Relative sparing of a thin rim of white matter, corresponding to the arcuate fibers, was noted between the cortex and the demyelinated foci (Fig 2). Other similar lesions were identified in the ventrolateral nuclei of the thalami, basal ganglia, and external and internal capsules. All extrapontine lesions were histologically of similar age to those in the pons.

Discussion

The osmotic demyelination syndrome is characterized by rapidly evolving corticospinal and bulbar dysfunction, often developing after rapid correction of hyponatremia (1). The clinical presentation typically includes acute changes in mental status, progressive spastic quadriplegia, and pseudobulbar palsy frequently leading to coma and death. Osmotic demyelination syndrome was suggested by Sterns et al (1) in patients who had both central pontine and extrapontine foci of myelinolysis. Gocht and Colmant (2) reported extrapontine myelinolysis in 53% of 58 autopsies of patients with syndrome and identified the areas most frequently involved as the cerebellum, lateral geniculate bodies, external and extreme capsules, and subcortical white matter.

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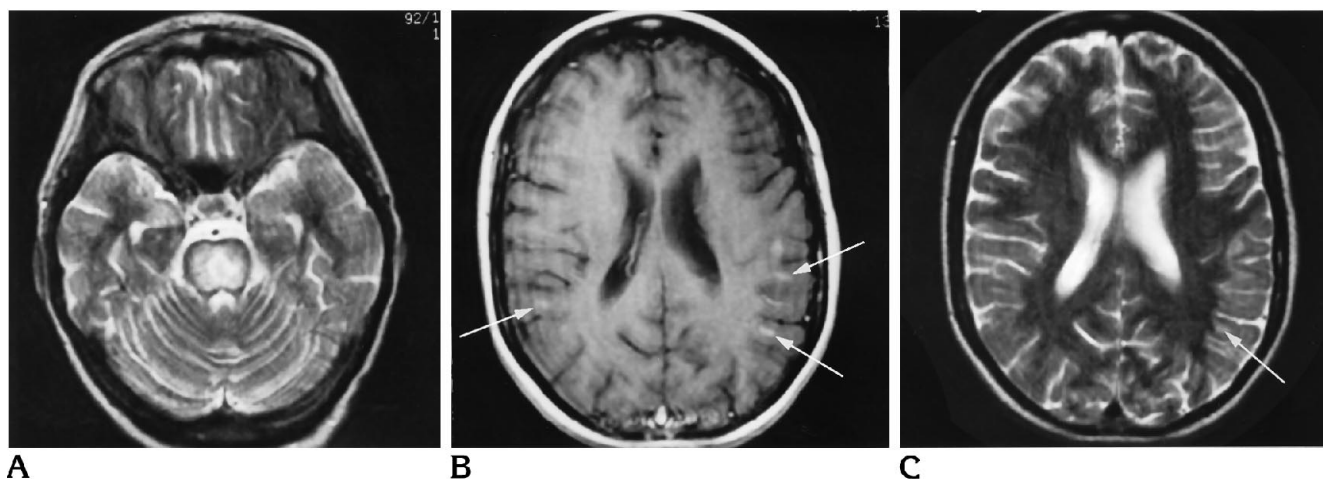


Fig 1. A, Axial T2-weighted MR image (3000/102/2 [repetition time/echo time/excitations]) shows a hyperintense lesion in the central portion of the pons with sparing of the periphery.

B, Axial T1-weighted image (500/20/2) after injection of contrast material shows enhancing lesions in the subcortical white matter of the right and left parietal lobes (arrows).

C, Axial T2-weighted image (3000/102) obtained at the same level as B shows a small linear area of increased signal intensity in the subcortical white matter (arrow).

Norenberg (3) hypothesized that during too rapid or excessive rise in serum sodium from a hyponatremic state, osmotic endothelial injury probably occurs with release of myelinotoxic factors and demyelination. The myelinotoxic factors may derive from the highly vascular gray matter and interact with adjacent myelin-containing white matter. Although this theory would explain the predominance of lesions in areas of gray and white matter apposition such

as the pons and basal ganglia, the presence of some intact white matter between the cortex and the subcortical demyelinated foci in our patient seems to contradict it.

MR is the technique of choice in imaging osmotic demyelination syndrome because of its ability to display the brain stem without hindrance from beam-hardening artifacts and its particular sensitivity to white matter changes. The central involvement of the pons with sparing of the periphery in our patient is identical to that described by Miller et al (4) in 13 patients with the syndrome.

In our patient, the diffuse ovoid or streaky subcortical lesions with their long axis parallel to the gyral surface were poorly seen on proton density- and T2-weighted images because of their small size and lack of distinctiveness from adjacent subarachnoid space. They were shown well on the enhanced study, likely because of the transient breakdown of the blood-brain barrier, as was described in the pons by Koch and Smith (5).

In conclusion, subcortical white matter lesions, in addition to the more typical pontine lesions, can be present in osmotic demyelination syndrome, and their demonstration should be attempted with enhanced MR. The association of pontine and other enhancing subcortical lesions is very suggestive of the syndrome, particularly after rapid correction of hyponatremia.

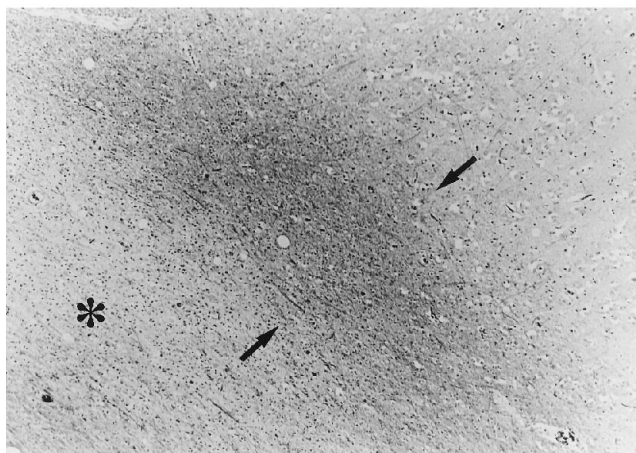


Fig 2. A section from the occipital lobe (myelin stain, original magnification $\times 80$) shows normal cortex in the upper right, with an area of white matter demyelination in the lower left (asterisk). Between the cortex and the lesion is a thin band of normal white matter (arrows).

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