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Case Report

Osmotic Demyelination Syndrome in Chronic Alcoholism: A Case Report

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Abstract

Osmotic demyelination syndrome corresponds to demyelination of the central part of the protuberance and may localize elsewhere. The main risk factors are chronic alcoholism, undernutrition and too rapid correction of hyponatremia. Symptoms vary depending on the location of the lesions. The classic presentation is tetra-paresis, or even locked-in syndrome, with abnormal movements and psychiatric manifestations occurring a few days after correction of hyponatremia. The diagnosis is confirmed by magnetic resonance imaging, which shows a T1-weighted hypointense and a hyperintense on T2-weighted and FLAIR sequences. To date, there is no treatment available, and only symptomatic therapy can be proposed.

Keywords: *Osmotic Demyelination Syndromes; Central Pontine Myelinolysis; Extrapontine Myelinolysis; Chronic Ethylism*

Introduction

Congenital osmotic demyelination syndrome is a rare and severe neurological disorder characterized by non-inflammatory demyelination of the central nervous system, predominantly affecting the pons and potentially other brain regions. It classically occurs after overly rapid correction of chronic hyponatremia, but is also associated with chronic alcoholism and malnutrition.

Clinical presentation is variable and often delayed, which may complicate diagnosis. Imaging, particularly brain magnetic resonance imaging (MRI), plays a key role in demonstrating characteristic lesions with hyperintensity T2 and FLAIR and hypointensity T1. We report a case highlighting the crucial role of MRI in the diagnosis of this condition.

Case Report

This is a 41-year-old patient presenting with ataxia, gait disturbance and paresthesia of the lower limbs, with a history of chronic alcoholism. The clinical examination reveals a patient with an altered general condition, but the blood tests shows normal sodium levels. A cerebral CT scan was ordered (Figure 1A), showing a cerebral pontine middle hypodensity without pathological contrast. MRI was performed (Figure 1B), which showed the middle cerebral pontine lesion in hypointense T1 weighted, hyperintense T2 weighted and Flair without contrast enhancement.

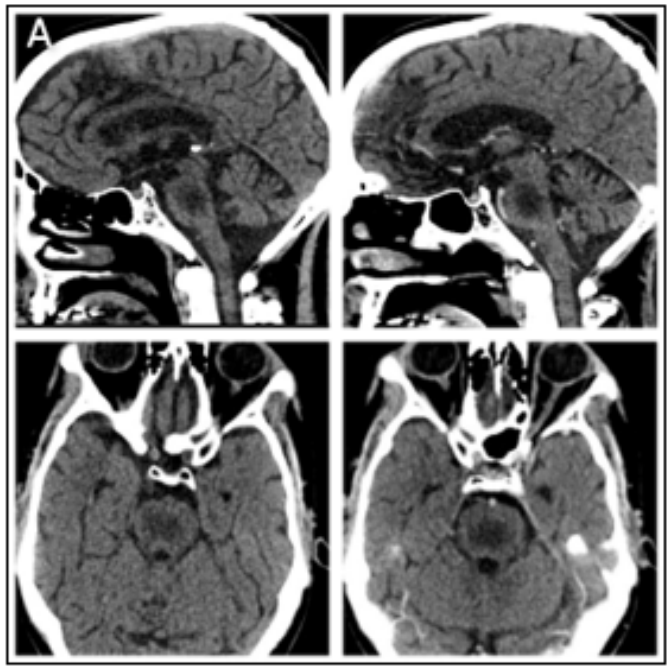

Figure 1A: *Sagittal and axial CT images with and without contrast showed a hypodense lesion in the midbrain without pathological contrast enhancement.*

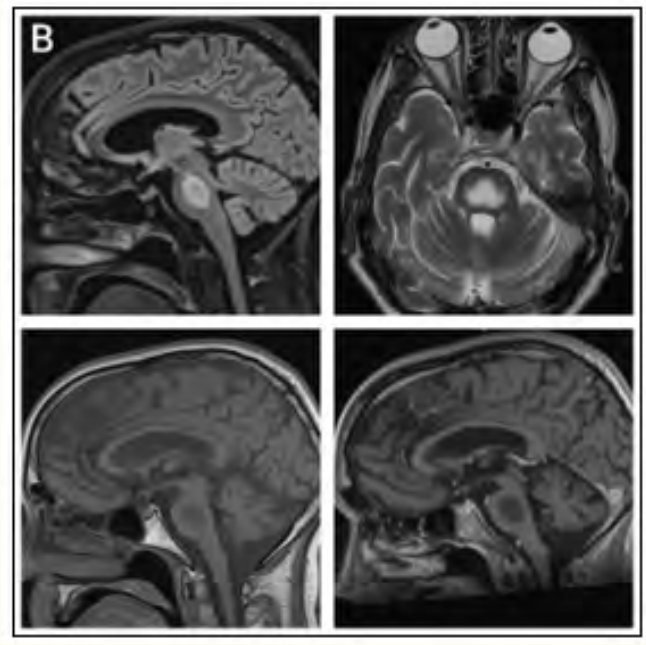

Figure 1B: *Sagittal and axial brain MRI with and without contrast injection showing the midbrain lesion with hypointense T1 weighted, hyperintense T2 weighted and Flair without contrast enhancement.*

Discussion

Osmotic demyelination syndromes are a rare pathology, grouping together central pontine and extrapontine myelinolysis, which share the same pathophysiology and risk factors, but differ in their clinical presentation, their association is often frequent (30% of cases).

It is defined by symmetrical destruction of myelin in all nerve fascicles, associated with loss of oligodendrocytes.

It occurs as a consequence of chronic ethylism and malnutrition, or too rapid correction of chronic hyponatremia.

Clinically, central pontine myelinolysis is suggested by disorders of consciousness or vigilance consciousness or vigilance, oculomotricity disorders, tetraparesis and even tetraparesis, or even a pseudobulbar syndrome or deep coma, reflecting the extent of demyelination.

Whereas in the case of extrapontine myelinolysis, extrapyramidal signs (parkinsonian syndrome, dystonia), behavioral disorders and convulsions.

This symptomatology follows a biphasic evolution: initially, the patient presents an encephalopathy which may recover rapidly, followed by the appearance of dysarthria and dysphagia secondary to cortico-bulbar damage, and flaccid quadriparesis which then becomes spastic secondary to cortico-spinal damage.

The positive diagnosis is often clinical, and should be made in the presence of any acute neurological syndrome in an alcohol-dependent, malnourished patient.

Computed tomography (CT) is not very sensitive, and may reveal a hypodense medio-protuberential area not enhanced by contrast medium.

Cerebral MRI remains the radiological examination of choice, demonstrating protuberance lesions in the form of a hyperintense on T2-weighted and FLAIR sequences, and a hypointense on T1-weighted sequences.

These signal anomalies spare the cortico-spinal fascicles and the ventro-lateral regions of the bridge. Other areas of the central nervous system may be affected, defining extrapontine myelinolysis, notably the cerebellum, thalamus, basal ganglia, cerebral cortex and subcortical white matter. The onset of these abnormalities is usually delayed by 10 to 15 days from the onset of clinical symptoms.

There is no correlation between the size of lesions and the severity of initial neurological damage, just as there is no correlation between the evolution of radiological abnormalities and the persistence of symptoms.

Cerebrospinal fluid studies are usually unremarkable, but hyper proteinorachia may be present. The EEG, which is non-specific, usually shows a diffuse slowing of brain activity.

At present, no curative treatment has been validated, so management is based on symptomatic and preventive treatment, based on reasoned correction of any hyponatremia, without exceeding a correction rate of 0.5 mmol/l per hour, with multi-day monitoring of natremia if necessary.

Although this syndrome is not irreversible, the prognosis remains guarded, with an often-unfavorable evolution and high mortality. The survival rate is estimated at 5 - 10% [1-5].

Conclusion

Osmotic demyelination syndrome is a demyelinating disorder of the central nervous system defined by clinico radiological arguments in an etiological context likely to orient the diagnosis, in particular a rapid correction of hyponatremia or chronic alcoholism. MRI remains the examination of choice for making a positive diagnosis.

The course is often unfavorable, with a guarded prognosis. Treatment remains symptomatic and preventive, with avoidance of etiological factors, particularly toxic ones, chronic alcohol abuse, and over-rapid correction of hydroelectrolytic disorders.

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Conflict of Interest

The authors declare no conflict of interest.

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