Case Report

Wernicke Encephalopathy and Central Pontine Myelinolysis: An Underdiagnosed Combination in Alcoholics

Amparo LOPEZ-BERNUS¹, Alvaro MUÑOZ-GALINDO³, Maria Teresa MOREIRO-BARROSO¹, Virginia VELASCO-TIRADO¹, Adela CARPIO-PEREZ¹, Moncef BELHASSEN-GARCIA²

¹Complejo Asistencial Universitario de Salamanca (CAUSA), Servicio de Medicina Interna, Salamanca, España ²Universidad de Salamanca, IBSAL, Salamanca, España ³Complejo Asistencial Universitario de Salamanca (CAUSA), Medicina Comunitaria, Salamanca, España

Summary

Wernicke’s encephalopathy (WE) and central pontine myelinolysis (CPM) are two neurologic diseases associated with several conditions. In patients with chronic alcoholism the combination of WE and CPM is rare and little is known about its clinical picture and the radiological changes. We report a chronic alcoholic patient, who had classic clinical features of WE with additional features of CPM without hyponatremia during alcohol withdrawal. The symptoms gradually improved after 1 month with only conservative treatment, and the 4 month-follow-up MRI showed complete resolution of the pontine lesions.

Key words: Alcohol-related problems; MRI; Clinical neurology

INTRODUCTION

Wernicke’s encephalopathy (WE) is a serious but curable neurologic disease caused by thiamine (vitamin B1) deficiency. It is unclear how thiamine deficiency causes brain lesions. Because of its role in cerebral energy utilization, it has been proposed that its deficiency initiates neuronal injury by inhibiting metabolism in brain regions with high metabolic requirements. WE is often associated with...
chronic alcoholism, poor dietary intake, increased metabolic requirements, hyperemesis of pregnancy and renal dialysis. The classic triad of WE includes consciousness changes, oculomotor dysfunction and ataxic gait\(^8\). WE is primarily a clinical diagnosis; the biggest barrier to diagnosis is a low index of suspicion, given that all features of the triad were recognized in only one-third of patients; in most cases, elements of the clinical triad appeared alone or in combination. Laboratory studies and neuroimaging studies can be helpful. Prognosis is improved by prompt administration of thiamine. Untreated WE leads to coma and death.

Central pontine myelinolysis (CPM) is an osmotic demyelination syndrome (ODS). The etiology of CPM is unclear, although rapid fluctuations of serum sodium levels in severe hyponatremia (serum sodium concentration is almost always 120 mEq/L or less) have been suggested as the main cause. CPM is most often found in patients with chronic alcoholism, malnutrition, hyponatremia, liver disease, liver transplants, systemic hypotension and infections. The clinical manifestations are typically delayed for two to six days after overly rapid elevation of the serum sodium concentration. Clinical manifestations are extremely diverse ranging from asymptomatic course, mild tremor or dysarthria to classic locked-in syndrome. Magnetic resonance imaging (MRI) plays an essential role in determining the number and extension of the lesion\(^2\). The prognosis of CPM can be highly heterogeneous, ranging from complete recovery to progression and death\(^9,12,17\).

Although coexistence of WE and CPM has been reported in some specific conditions, in patients with chronic alcoholism the combination of WE and CPM is considered a rare condition and little is known about its clinical picture and the radiological changes\(^20\). We presented a chronic alcoholic patient with a WE and CPM without hyponatremia during alcohol withdrawal with a good recovery.

**CASE PRESENTATION**

A 50-year-old man developed symptoms of 15 days of evolution with asthenia, difficulty walking and decreased visual acuity. He has depressive syndrome treated with escitalopram (10 mg/day) and chronic alcohol consumption of 6-8 SDU/day (he reports not having drunk alcohol in the last week). Physical examination shows bradypsychia and attention deficit, right lateral rectus palsy and symmetrical ataxic gait. The laboratory analyses show AST 86 IU/L, ALT 30 IU/L, GGT 79 IU/L. Complete blood count, coagulation and ionogram are within normal limits (Natremia 137 mEq/L) and the urine test for toxic substances is negative. The brain CT scan reveals signs of cerebral and cerebellar atrophy. The cerebrospinal fluid does not present relevant alterations. WE is suspected and a treatment with intravenous thiamine is started (300 mg/day), 0.9% saline and 10% dextrose solution and supportive treatment. Afterwards, the patient presents tonic-clonic seizures that are attributed to alcohol withdrawal syndrome and which disappear with intravenous diazepam (10 mg). The patient recovers his level of consciousness and the ocular involvement also disappears, although the ataxic gait is still present. One week later he presents new signs of decreased consciousness, with generalized hyperreflexia and significant spasticity in all four limbs. He does not present significant metabolic alterations, and the natremia remains stable between 136 and 145 mEq/L. The cerebral magnetic resonance imaging (MRI) reveals hyperintense T2-weighted sequences and local central flair sequences in the pons. These findings are compatible with central pontine myelinolysis (Fig. 1). After one week of supportive treatment, the patient shows an improvement of his level of consciousness, hyperreflexia, spasticity and ataxic gait. In the control tests carried
out after 4 months he is asymptomatic and the MRI is normal (Fig. 2).

**DISCUSSION**

We reported the clinico-radiological course in an alcoholic patient with CPM and WE. WE is an acute, neuropsychiatric syndrome, characterized by nystagmus and/or ophthalmoplegia, mental status changes, and ataxic gait\(^{(19)}\). Our patient met the four Caine criteria for diagnosis of WE: dietary deficiency, oculomotor

---

*Figure 1: Local central hyperintense areas in the pons. Findings compatible with central pontine myelinolysis.*

*Figure 2: Complete resolution of the lesions in the pons 4 months later.*
abnormalities, cerebellar dysfunction and altered mental status. Acute WE lesions are characterized by vascular congestion, microglial proliferation, and petechial hemorrhages. In chronic cases, there is demyelination, gliosis, and loss of neuropil with relative preservation of neurons. While most often associated with chronic alcoholism, WE occurs also in the setting of poor nutrition caused by malabsorption, poor dietary intake, increased metabolic requirement (e.g., during systemic illnesses), or increased loss of the water-soluble vitamin thiamine. Signs and symptoms associated with thiamine deficiency lack sensitivity and specificity, and hence thiamine deficiency is frequently underdiagnosed by physicians. In our cases, the neuroimaging tests were negatives, and we could not rule out the diagnosis of WE, because abnormalities on CT or MRI have only been reported in a small number of patients with WE with a characteristic, symmetrical distribution in structures surrounding the third ventricle, aqueduct, and fourth ventricle. MRI is more sensitive than CT in detecting acute lesions (53% vs 13%)1. Given the fact that a delay in treatment worsens prognosis, if WE is suspected, treatment with thiamine, intravenously or intramuscularly, should be initiated immediately, and continued until a normal diet is resumed. The rapid improvement in ocular signs after thiamine administration emphasizes the role of WE in our patient.

CPM is a rare but devastating cause of morbidity and mortality, due to demyelination of the pons. On rare occasions, demyelination occurs outside the pons7. CPM is characterized by non-inflammatory, frequently symmetrical loss of myelin in the basis pontis, with preservation of axons and neuronal cell bodies. The precise mechanism by which cellular injury occurs in CPM is unknown, it is most likely multifactorial in nature and the underlying chronic diseases may be central to the development. Previous clinical and laboratory studies have suggested that osmotic vascular endothelial injury is a pathogenic mechanism for osmotic demyelination syndrome (ODS) and this causes the release of myelotoxic factors, production of vasogenic edema and a resulting injury to oligodendrocytes4,16. ODS has also been reported in cases without significant shifts of the serum sodium, and with gradual correction of hyponatremia4,5,13,14. Chronic alcoholism is the most common predisposing condition for developing ODS; in cases associated with chronic alcoholism, many patients developed ODS during the terminal stage of binge drinking, yet there have been few case reports of ODS after alcohol withdrawal10,21, and the majority of cases of ODS with normonatremia were associated with chronic alcoholism15. The brain lesions associated with ODS can be occasionally detected by CT scanning or, much more reliably, by MRI. An initially negative radiologic study in a patient who develops neurologic symptoms after overly rapid correction of hyponatremia does not exclude ODS. In CPM without hyponatremia there is no specific treatment, but major fluctuations in serum osmolality should be avoided as clinical and radiological recovery from CPM is possible4. Given the potentially severe and permanent adverse consequences of ODS, prevention is essential. In our case the symptoms of the patient gradually improved after 1 month with only supportive treatment. ODS is actually compatible with good recovery in more than half of cases18.

The combined clinical manifestations are infrequent, although previous postmortem studies suggested that pathological lesions of WE were found in 30-40% of CPM patients3,6. A possible pathogenic explanation of this coexistence could be the fact that the interrelation of thiamine metabolism and serum osmotic variation may be involved in frequent complications of CPM and WE. Previous thiamine
deficiency has been thought to prompt osmotic damage to the central pons in non-hyponatremic patients with alcoholic WE\(^8\). Coexistence of WE and CPM has been reported in some specific conditions like parenteral nutrition or hyperemesis gravidarum\(^{20}\). In patients with chronic alcoholism, the combination of WE and CPM is a rare entity. A possible explanation could be infradiagnosis, due, among other reasons, to the lack of clinicoradiologic specific data, as in the case of our patient. In spite of the severity of the symptoms, our patient had a good recovery with only conservative treatment.

**Conflict of interest**

All authors declare no potential conflicts of interest and no sources of support.

**Correspondence to:**

Moncef Belhassen-garcia  
E-mail: mbelhassen@hotmail.com

---

**REFERENCES**


