Central pontine myelinolysis in a chronic alcoholic: A clinical and brain magnetic resonance imaging follow-up

Centralna pontina mijelinoliza kod hroničnog alkoholizma: klinički oporavak uz postojanu leziju na magnetnoj rezonanci mozga

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Abstract

Introduction. Central pontine myelinolysis (CPM) is a noninflammatory, demyelinating lesion usually localised in the basis pontis. Chronic alcoholism is frequently associated with this condition which may have a variable clinical outcome. Until now, brain magnetic resonance imaging (MRI) follow-up in alcoholic CPM cases after alcohol withdrawal has been rarely described. Case report. We reported a 30-year-old male with a 12-year history of alcohol abuse, who presented with inability to stand and walk, nausea, vomiting and somnolence. Neurological examination revealed: impaired fixation on lateral gaze, dysarthria, mild spastic quadriparesis, truncal and extremity ataxia, sock-like hypesthesia and moderate decrease in vibration sense in legs. Brain MRI showed a trident-shaped non-enhancing pontine lesion highly suggestive of CPM. After an eight-month alcohol-free follow-up period, the patient’s clinical status significantly improved, while the extent of MRI pontine lesion was merely slightly reduced. Conclusion. The presented case demonstrates that CPM in chronic alcoholics may have a benign clinical course after alcohol withdrawal, which is not necessarily associated with the reduction of lesions on brain MRI.

Key words: demyelinating diseases; pons; diagnosis; magnetic resonance imaging; alcoholism; treatment outcome.

Introduction

Central pontine myelinolysis (CPM) is a noninflammatory, demyelinating lesion usually localised in the basis pontis. CPM has been reported to affect patients with a history of chronic alcoholism, malnutrition, dysionemia or rapid correction of hyponatriemia. Before the introduction of magnetic resonance imaging (MRI), the diagnosis of CPM was frequently established postmortem, so the prognosis of this condition was doubtful, but nowadays rare asymptomatic and benign CPM cases have been reported. However, longitudinal brain MRI findings in alcoholic CPM patients following alcohol withdrawal are scarce.

Apstrakt

Uvod. Centralna pontina mijelinoliza (CPM) je neinflamatarno i demijelinizaciono oštećenje ponsa koje se može javiti kod hroničnog alkoholizma. Prognoza CPM je varijabilna, a prikazi nalaza na magnetnoj rezonanci (MR) mozga boleznih sa CPM kod alkoholizma koji su prospektivno pružili nakon alkoholne apstinencije retki su u literaturi. Prikaz bolesnika. Prikazan je 30-godišnji bolesnik koji je konzumirao prekomerne količine alkohola tokom 12 godina, kod koga su se subakutno ispoljile nemogućnost samostalnog stajanja i hoda, mučnina, povraćanje i pospanost. U neurološkom nalazu postojali su slabost fiksacije pri pogledu u levo i desno, dizartrijija, znaci blage spastične kvadruparesis, blaga do umerena trunkalna ataksija, hipestezija za površni dodir poput čarapa i skraćen vibracijski senzibilitet na nogama. Na MR mozga utvrđena je lezija oblika trozupca u centralnom delu ponsa koja je imala karakteristike CPM. Nakon osam meseci alkoholne apstinencije klinički neurološki status kod bolesnika značajno se popravio, dok je kontrolnim MR pregledom mozga pokazana samo minimalna regresija ranije verifikovane ekstenzivne pontine lezije. Zaključak. CPM kod hroničnog alkoholizma može imati povoljan klinički tok i prognozu nakon prestanka konzumiranja alkohola uprkos održavanju lezije na MR mozga.

Ključne reči: demijelinizacione bolesti; pons; dijagnoza; magnetna rezonanca, snimanje; alkoholizam; lečenje, ishod.
Case report

A 30-year old male with a 12-year history of chronic alcohol abuse, who presented with inability to stand and walk, nausea, vomiting and slight somnolence, consumed up to 2.5 L of home-made brandy per day over the year preceding the onset of neurological manifestations. Physical examination was normal. Neurological examination revealed the following: impaired fixation on lateral gaze, dysarthria, mild bilateral upper and lower extremity weakness with slightly exaggerated tendon reflexes on upper extremities and diminished tendon reflexes on lower extremities; mild upper limb and moderate lower limb ataxia, moderate truncal ataxia; sock-like hypesthesia and moderate decrease in vibration in legs. The patient walked with unilateral assistance. Brain MRI performed at that time revealed a trident-shaped pontine abnormality on axial images (or omega sign) that presented as a non-enhancing T1-weighted hypointensity (Figure 1A) and T2-weighted hyperintensity (Figure 1B) which spared the outer rim of the pons. No other brain MRI abnormalities were detected. At that time, the patient refused hospitalization, as well as the participation in any additional diagnostic procedure including blood withdrawal.

Two months after the initial presentation, the patient joined the alcoholism rehabilitation program and his neurological status gradually improved without any specific treatment. After an eight-month alcohol-free period and eleven months after the initial presentation, a follow-up examination revealed normal findings on cranial nerves, no extremity weakness and slightly exaggerated tendon reflexes on upper extremities; a minor upper extremity ataxia which patient was not aware of was noticed, as well as a mild lower limb ataxia, positive Romberg test with the eyes closed and normal walking. Sensory impairment was still present unchanged. At that time the patient accepted to have blood withdrawn for hematology and biochemistry panel, including blood sodium and potassium levels, and laboratory findings were normal. A follow-up MRI showed the persisting trident-shaped pontine lesion whose volume was slightly reduced compared to the baseline scan performed eleven months earlier (Figure 1 C, D).

Discussion

We reported a CPM case in a chronic alcoholic with a significant clinical recovery after alcohol withdrawal, not associated with a significant resolution of a pontine MRI lesion.
Brain MRI in our patient showed a trident-shaped symmetrical non-enhancing central pontine abnormality which suggested CPM. Although, a typical CPM lesion usually spares ventrolateral longitudinal fibres and corticospinal tracts, our patient had a mild spastic quadriparesis at the initial presentation. The reported patient did not have extrapontine brain MRI lesions. However, until now, symmetrical foci of extrapontine myelinolysis (EPM) have been frequently reported in other brain regions (in descending order of frequency: cerebellum, lateral geniculate body, external capsule, extreme capsule, hippocampus, putamen, cerebral cortex/subcortex, thalamus, caudate nuclei, caustreum, internal capsule, midbrain, internal medullary lamella, mammillary body, medulla oblongata).

In line with the MRI findings in our patient, both CPM and EPM typically present with a non-enhancing hypointensity on T1-weighted brain MRI images, hyperintensity on T2-weighted and fluid attenuated inversion recovery sequences. Additionally, in certain CPM/EPM cases, contrast-enhancement of T1-weighted brain MRI lesions was described in early phases up to 4 weeks after the initial clinical manifestation and diffusion-weighted imaging changes were reported before the development of conventional MRI signal intensity abnormalities.

CPM and EPM referred as osmotic demyelination syndromes (ODMS) have been shown to share similar histology. The pathophysiology of ODMS is poorly understood, but the proposed mechanisms involved in its development include osmotic injury to the vascular endothelial cells, vasogenic edema and/or brain dehydration, the release of myelinotoxic factors, as well as axonal separation from the myelin sheath which contributes to oligodendrocyte injury and demyelination. It has been shown that edema and/or demyelination in ODMS could be reversible, potentially correlating with a resolution of brain MRI lesions, which varies from their complete disappearance to merely slight reduction on conventional MRI. Residual MRI findings have been considered to represent areas of permanent damage.

Chronic alcoholism is a common predisposing condition for the development of ODMS. Although alcohol itself could interfere with sodium/water regulation by suppression of antidiuretic hormone, an inadequate nutrition/water intake and/or liver dysfunction in alcoholics might also contribute to ODMS. Additionally, chronic alcoholics might not be able to maintain protective cerebral mechanisms against osmotic stress, and could also suffer from direct toxicity of alcohol. Furthermore, oxidative stress in alcoholics may favor apoptosis of brain cells, leading to an irreversible damage.

However, it has been suggested that a better outcome of CPM/EPM could occur in chronic alcoholics in which it may be asymptomatic or have relatively few symptoms, than in cases associated with an acute correction of hyponatremia in which a high mortality rate was reported with a significant neurological deficits in the survivors. Clinical recovery of our patient was associated with the alcohol withdrawal without any other specific treatment. Since he did not accept any diagnostic tests in the acute phase of his illness, apart from brain MRI, we can only speculate that CPM in this case developed due to alcohol-related mechanisms and not as a consequence of disturbances in blood sodium level.

Clinical recovery which was not accompanied by a significant resolution of MRI lesions in our patient supports the notion that follow-up MRI in CPM does not have a significant prognostic value for the outcome of this disorder.

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