Central Pontine Myelinolysis Due to Chronic Alcohol Use: Case Report

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Abstract

Central pontine myelinolysis (CPM) is a neurological disorder characterized by demyelination on the bottom of pons. CPM is known to be the most common clinical presentation of osmotic demyelination syndrome. Osmotic stress formed by rapid correction of hyponatremia in glia cells is thought to be important in pathogenesis. Oligodendroglias are more sensitive to dehydration and volume changes as they are tightly aligned in the pontine. Chronic alcohol use is a rare cause of osmotic demyelination. In chronic alcoholics, central pontine myelinolysis may be asymptomatic or mild symptoms may develop. We presented the case to emphasize that chronic alcoholism is a rare cause of central pontine myelinosis.

Keywords: myelinolysis, central pontine, alcoholism.

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INTRODUCTION

Osmotic demyelination syndrome (ODS) was first described in 1959 [(1)]. Central pontine myelinolysis is a neurological condition which is characterized by demyelination in the pontine and extrapontine, amygdaloid nucleus, subthalamic nucleus, corpus geniculatum laterally, thalamus, striatum, and cerebellum. Although it is an important risk factor ODS, rapid correction of hyponatremia has been reported in normonatremic and hypernatremic patients. It is common with in particular, chronic alcoholism, liver transplantation, hypokalemia, diabetes mellitus, hepatocellular dysfunction, chemotherapy, lithium toxicity and chronic renal failure [(2-4)].

The mechanisms that cause osmotic demyelination syndrome are not fully known. The most important reason is that cytotoxic plasma components pass the blood brain barrier as a result of osmotic changes in endothelial cells. The co-occurrence of these events induces astrocytic apoptosis, disrupts the myelin structure of oligodendrocytes and causes the release of inflammatory cytokines, leading to the activation of microglia [(5-6)]. Hyponatremia creates intracellular volume changes, thereby the soluble substances in the cell are redistributed and show osmotic adaptation, the body is protected from the cerebral edema, but as a result, the intracellular organic

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osmotic active particles are reduced, in five days, the repositories of osmotic active particles are formed again.

The osmolality of the serum shows a rapid increase and the brain is at risk of osmotic dehydration [7]. The hypertonic fluid, which causes edema in classical osmotic demyelination syndrome, is present in the extracellular space until endothelial integrity is achieved and this liquid shows its toxic effects on myelin and oligodendrocytes. Pathological mechanism in osmotic demyelination syndrome is a change in the level of abrupt plasma solutes. Osmotic damage to the vascular endothelial cells and this cause, the release of myelinotoxic factors, the cause of vasogenic edema and/or brain dehydration.

The three important molecules with osmotic gradient are BUN, sodium and glucose. Changes between them cause cerebral edema [8]. The mechanisms of CPM and EPM are poorly understood. Chronic alcoholics may not be able to sustain preventive cerebral mechanisms against osmotic stress, as well as suffering from the direct toxicity of alcohol, the extreme production of free radicals and the deranged nitric oxide metabolic effects in alcoholics may favor apoptosis of brain neurons. We presented this case to emphasize the importance of diagnosis of CPM due to chronic alcohol use.

CASE REPORT/CASE PRESENTATION

A thirty-eight-year-old male patient was admitted to the emergency department because of a speech disorder, weakness in the left arm and leg that started one day ago. In his neurological examination, as pathological finding, dysarthria, left 4/5 hemiparesis and bilateral extensor plantar response was determined. He had a history of chronic alcohol use for about twenty years. The routine blood tests revealed AST: 139, ALT: 114, LDH: 269, GGT: 1155, no pathological findings were found in hormone and hemogram examinations.

Axial brain tomography (CT) (Fig. 1) showed a hypodense appearance in the pontine. In Cranial Magnetic Resonance (MR) images; Axial T2A- weighted (Fig. 2) image had an extensive hyperintense appearance that did not contain anterior segment in pontine. Diffusion-weighted (Fig. 3) image showed extensive hyperintense signal changes in which the anterolateral section (pyramidal tract) was preserved in the pontine. Axial

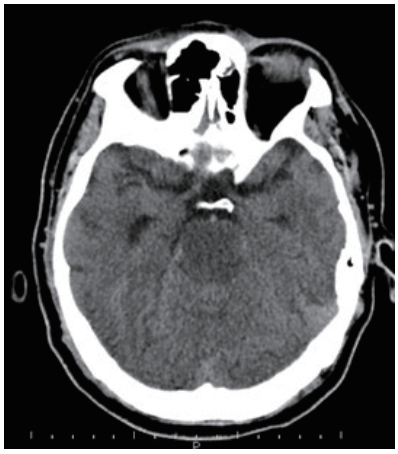

Figure 1. Axial Brain tomography (CT), the image showed hypodense appearance in the pontine.

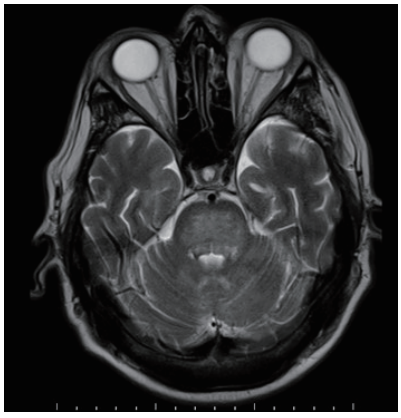

Figure 2. In Axial T2A -weighted image, extensive hyperintense appearance that does not contain anterior segment in the pontine.

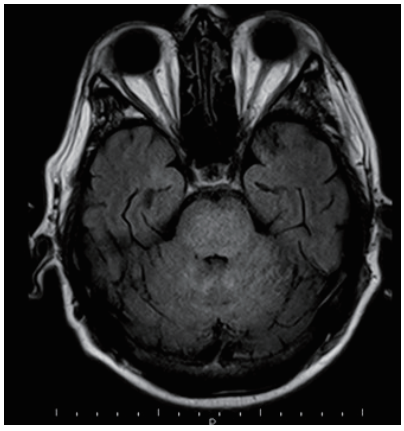

Figure 3. Diffusion image, extensive hyperintense signal changes in which an anterolateral segment (pyramidal tract) is preserved

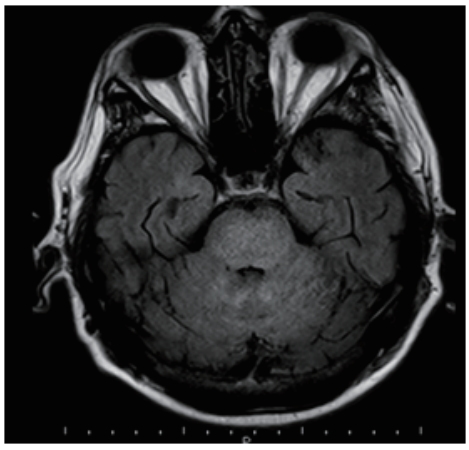

Figure 4. Axial FLAIR image showed extensive hyperintense signal changes in the pontine..

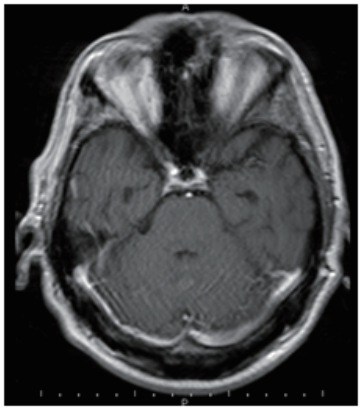

Figure 5. Contrast-enhanced Axial T1- weighted image showed no contrast enhancement in the pontine.

FLAIR (Fig. 4) image showed extensive hyperintense signal changes in the potine. In contrast-enhanced T1-weighted image, no contrast enhancement was detected (Fig. 5).

DISCUSSION

The mechanism that causes osmotic demyelination syndrome is not known exactly and there is no specific treatment. Once diagnosed, treatment is supportive and treatment should be done to prevent complications. Reports from randomly selected cases and small case series; steroid, intravenous immunoglobulin and thyroid-releasing hormone, re-induction of hyponatremia, application of organic osmoliths (urea, myoinositol), and dopaminergic compounds have shown favorable results, especially in extrapontine myelinosis cases. Since no trial has been performed on humans, these possible treatment methods are not yet recommended for ODS patients [9-10].

In a study of 34 ODS patients conducted by Abbott et al., the mortality rate was 6%, the rate of fully recovering patients was 30%, the rate of patients who did not fully healed, but had been living independently, 32%, and the rate of patients who were dependent was reported to be 32% [11]. In the literature evaluation of Martin and et al., mortality rate in ODS was %40-50 and %10-20 was noted to be taken to intensive care unit [12]. In the literature, the mortality rate varies on a large scale from 6% to 90%. Menger and Jörg reported that 40% of the patients recovered without showing any neurological sequelae [13]. Radiologically, the differential diagnosis includes bacilli perforated artery infarcts, demyelinating diseases, pontine gliomas and hypertensive encephalopathies. In Computed tomography ODS is defined as non-mass density decrease predominantly in the pons, thalamus and basal ganglia [14]. Magnetic resonance in the diagnosis is a more sensitive neuroimaging. Magnetic resonance imaging shows hypointense on T1-weighted images and hyperintense signal changes on T2-weighted images in demyelination areas [15-16]. Diffusion limitation in myelinolysis sites has been described since the onset of symptoms. However, it is also emphasized in some studies that diffusion findings may extend until 24 hours after the onset of the clinic [17]. Lesions typically do not show enhancement [18-19]. In our case, neuroradiological imaging studies were consistent with ODS. In this context, cranial magnetic resonance imaging revealed no lesion in the pons and mesencephalon, and the T2-weighted and FLAIR sequences caused a signal increase. In the diffusion-weighted images, mesencephalon and pontine diffusion restriction were detected in which corticospinal tracts and transverse pontine fibers were preserved. The protection of ventrolateral pontine and corticospinal tracts, as in our case, is diagnostic in ODS [18-20].

Rapid correction of hyponatremia is an important risk factor for ODS but ODS can be seen in patients with chronic alcohol use [1]. Chronic alcoholism is the common causative reason of CPM [21]. But very little cases of CPM following alcohol withdrawal have been reported [22-23].

Chronic alcoholism may not be able to generate

protective cerebral mechanisms against the osmotic stress. People who use chronic alcohol have reduced glucose uptake due to malnutrition and thiamine deficiency, and they may not have sufficient energy reserves to synthesize organic osmoles. In addition, Na + / K + ATPase pump activity cannot be maintained because there is insufficient energy in the cells [24].

In our case, clinical findings appeared on the basis of chronic alcohol use and normonatremic.

CONCLUSION

Central pontine myelinolysis is a rare disease. If neurological examination is present in patients with chronic alcohol use, diagnosis of central pontine myelinosis should be considered.

The most important step in these patients is the early diagnosis and the immediate start of supportive treatment. Thus, disease can be prevented before neurological deficit develops.

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