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CASE STUDY

Central pontine myelinosis following alcohol withdrawal: report of two cases

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Osmotic demyelination syndrome (ODS) is a non-inflammatory demyelination illness and it includes both central pontine myelinolysis (CPM) and extrapontine myelinosis (EPM). These neurological conditions can rarely develop after alcohol withdrawal. While the precise pathophysiology of EPM and CPM is still unknown, various studies indicate that vasogenic edema is a major element in EPM while cytotoxic edema is a key factor in CPM. Here we present two patients who were diagnosed with central pontine myelinolysis following alcohol withdrawal and showed improvement with steroid therapy. Further studies are needed to demonstrate the effectiveness of steroid treatment in these patients. This case is highlighted due to the rarity of the disease and the observed benefits of high-dose steroid therapy.

Keywords: central pontine myelinolysis, osmotic demyelination syndrome, extrapontine myelinosis, alcohol withdrawal, high-dose steroid therapy, case report

Introduction

Osmotic demyelination syndrome (ODS) is a non-inflammatory demyelination illness and it includes both central pontine myelinolysis (CPM) and extrapontine myelinosis (EPM). These neurological conditions can rarely develop after alcohol withdrawal. While the precise pathophysiology of EPM and CPM is still unknown, various studies indicate that vasogenic edema is a major element in EPM and cytotoxic edema is a key factor in CPM (1). Here we present two patients who were diagnosed with central pontine myelinolysis following alcohol withdrawal and showed improvement with steroid therapy.

Case report 1

A 22-year-old Indian male presented to our emergency department complaining of fatigue and generalized body weakness with yellow discoloration of the sclera for 5 days. He had one episode of vomiting and headache, there are no other significant neurological or medical complaints. There is no past history of medical comorbidities. He is a smoker, an alcoholic, and has a history of multiple drug abuse in the past. He started drinking about a year ago, and the last time he consumed alcohol was one day before admission.

Examination at the time of admission showed that the patient was conscious and oriented. The examination of his cranial nerves was normal. He had hypotonia and weakness in both upper limbs, with power 4/5 proximal and 2/5 distally with weak hand grip. Lower limb examination showed proximal power 2/5 and distal power 0/5 with bilateral foot drop. Except for the sluggish triceps reflex, all the other reflexes were absent. Plantar reflex showed no response bilaterally. Sensory examination revealed reduced pinprick in glove and stocking distribution and reduced joint position sense in both lower limbs up to the ankle. His gait could not be tested.

His initial lab investigations showed macrocytic anemia with high mean cell volume, liver function tests were

deranged, and gamma glutamyl transferase was very high (Table 1). Other lab investigations, including the thyroid function test, hepatitis serology, toxicology panel, and autoimmune workup, were negative. Cerebrospinal fluid (CSF) analysis was normal.

The nerve conduction study showed evidence of motor and sensory polyneuropathy, probably axonal, as motor potentials were absent in both lower limbs, including F-waves.

The patient was admitted with a case of alcoholic liver disease with subacute motor sensory polyneuropathy and was on conservative treatment.

Plain magnetic resonance imaging brain (MRI) was done on the sixth day of admission as the patient was confabulating and confused. It showed a hyperintense lesion in T1 with corresponding hyperintensity in T2, and FLAIR in the central pons, suggestive of osmotic demyelination syndrome (central pontine myelinolysis) (Figures 1–3).

Methylprednisolone 1000 mg daily for three days was given, along with supportive and symptomatic care. The patient showed significant improvement in terms of motor power, sensory function, sensory and cognitive function. At the time of discharge, the patient was fully conscious, oriented, and mobilizing without support.

Case report 2

A 51-year-old Indian national presented to our emergency department with generalized tonic-clonic convulsions. He is an alcoholic and nonsmoker. There is no past history of medical comorbidities. He had no past history of seizure disorder. Examination at the time of admission, the patient was confused, agitated, and delirious and was having bilateral postural tremors. Laboratory investigations showed mildly deranged Liver Function Test with very high GGT level (Table 1). All the other basic metabolic parameters, toxicology screen, and inflammatory markers were within normal range. His initial CT brain on admission was normal. He was admitted as a case of alcohol withdrawal convulsion and was treated for the same.

During the course of the hospital stay, there were no further convulsions but he was still delirious and examination revealed truncal, gait, and appendicular ataxia. There was no ophthalmoparesis. So magnetic resonance imaging of the brain was done which showed symmetrical high T2/FLAIR signal abnormality centrally in the pons with a "trident-like" appearance suggestive of central pontine myelinolysis (Figures 4–6).

He was started on methylprednisolone 1000 mg daily for 3 days, along with supportive and symptomatic care. The patient gradually started improving in terms of his cognitive function and ataxia. He was able to walk without support on discharge.

Discussion

Corticospinal tracts in the pons and midbrain are involved in these conditions-causing, spastic quadriparesis, and pseudobulbar palsy. Some typical manifestations of this syndrome include ataxia, quadriparesis, dysarthria, ophthalmoplegia, and progressive fatigue. It was later discovered that EPM is the term representing the condition caused by osmotic demyelination that involves the pons and multiple extrapontine sites. Both aforementioned conditions are included in the osmotic demyelination syndrome. Dialysis, advanced lymphoma liver failure and transplantation, cachexia, carcinoma, pellagra, severe bacterial infections, chronic alcoholism, and acute hemorrhagic pancreatitis, are among the comorbidities

TABLE 1 | Showing important biochemical parameters of patients 1 and 2.

S. no	Laboratory investigations	Patient 1	Patient 2
1	HB (g/dl)	12.3	11
2	Sodium (mmol/L)	133	138
3	Potassium (mmol/L)	4.4	3.5
4	Creatinine (umol/L)	56	106
5	SGOT (IU/L)	25	121
6	SGPT (U/L)	99	31
7	Alkaline phosphatase (IU/L)	157	153
8	Ammonia (umol/L)	37	36
9	GGT (IU/L)	1032	1429

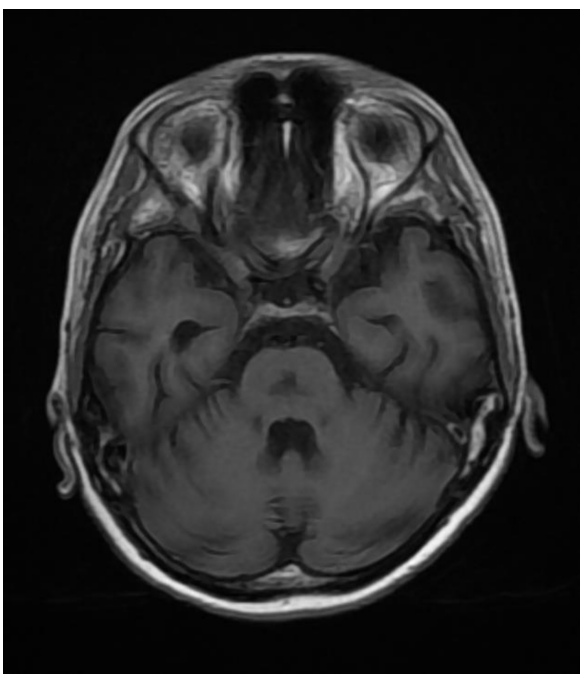

FIGURE 1 | Case report 1 (T1 weighted image shows hypointense lesion in central pons).

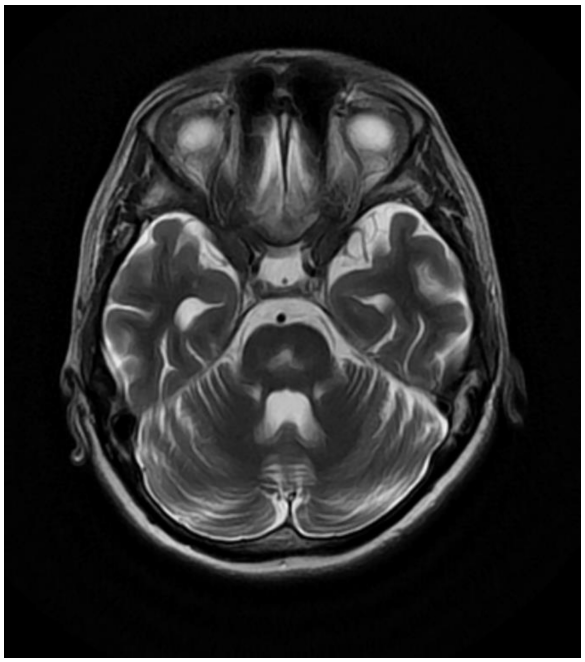

FIGURE 2 | Case report 1 (T2 weighted image shows hyperintense lesion in central pons).

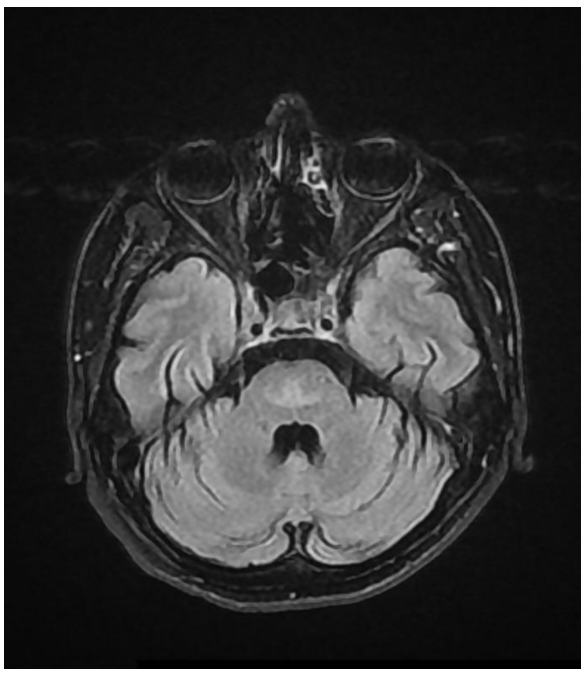

FIGURE 3 | Case report 1 (FLAIR image shows hyperintense lesion in central pons).

linked to an increased prevalence of CPM. When compared to CPM resulting from rapidly correcting hyponatremia, chronic alcoholism-associated CPM occurs in approximately 40% of patients and is typically benign and has a better prognosis (2).

Once clinical signs are identified, MRI of the brain is used to confirm the diagnosis of CPM (3). There is no relationship

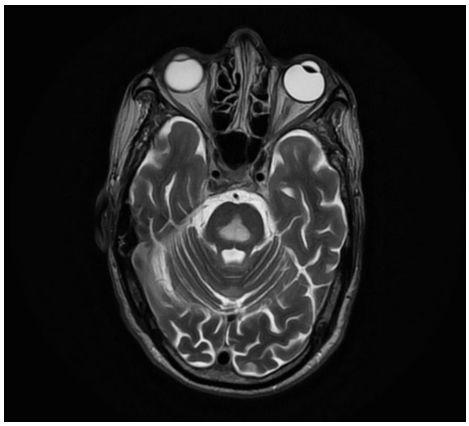

FIGURE 4 | Case report 2 (T2W image shows hyperintense signal in central pons).

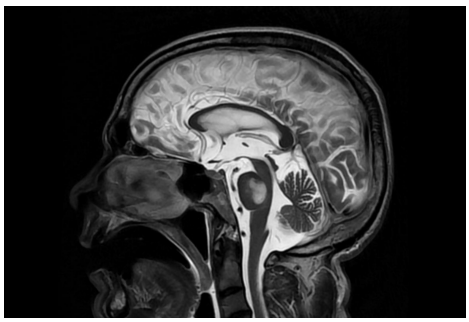

FIGURE 5 | Case report 2 (T2 sagittal image shows trident sign in central pons).

between the size of the pontine lesion and the severity of the neurologic condition or the outcome (4).

Norenberg suggests that individuals with chronic alcoholism might struggle to maintain protective cerebral mechanisms against osmotic stress while also experiencing direct toxicity from alcohol. Moreover, the excessive production of free radicals and disturbances in nitric oxide metabolism in alcoholics may contribute to the apoptosis of brain neurons. It is conceivable that the symptoms could be caused by the disruption of corticopontine networks, and an alternative hypothesis is that the interruption of neurotransmitter pathways originating from the brainstem, specifically the dopaminergic and cholinergic pathways, could have an impact on cognitive function (5).

In conclusion, chronic alcoholics may experience CPM and EPM as a result of alcohol withdrawal. CPM and EPM should be considered in addition to Wernicke's encephalopathy while evaluating patients with new psychotic and cognitive symptoms after alcohol withdrawal (6).

Even though the treatment for CPM is symptomatic and supportive care, there are few case reports (7) where high-dose steroids were used with significant improvement in clinical condition. Our patients received high-dose steroids for 3 days and showed significant improvement in cognitive

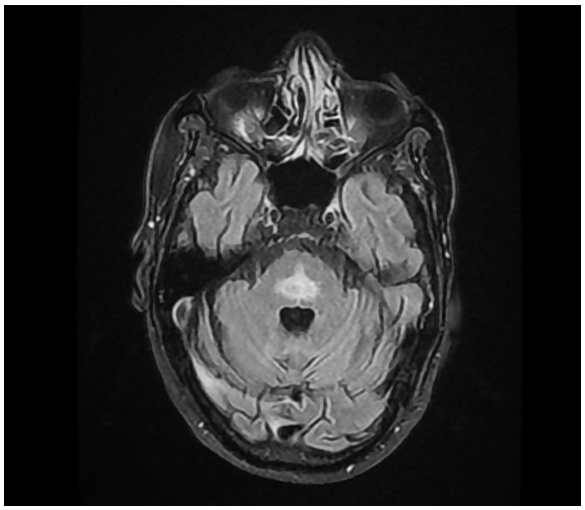

FIGURE 6 | Case report 2 (axial image shows hyperintense signal in central pons).

function and ataxia. Our first patient also had symptoms of neuropathy which improved after steroid therapy.

The majority of the cases of ODS with normonatremia were associated with chronic alcoholism. For the cases associated with chronic alcoholism, many patients developed ODS during the terminal stage of binge drinking. However, a small number of case studies have documented ODS occurring after alcohol withdrawal (8).

The cells may lack sufficient energy reserves to maintain the Na/K ATPase pump activity and to synthesize organic osmoles if the patient is malnourished, which is often the case in alcohol dependence cases. Due to its tendency to reduce brain glucose uptake, alcohol-associated thiamine deficiency may make the issue worse. A pro-apoptotic drive is the outcome of this imbalance in the energy supply and demand (9).

Conclusion

CPM and EPM can be observed in chronic alcoholics with normonatremia requiring a high index of suspicion to diagnose such cases. Our patients improved significantly with steroid treatment. Further studies are needed to demonstrate the effectiveness of steroid treatment in these patients. These case presentations are highlighted due to the rarity of the disease and the observed benefits of high-dose steroid therapy.

Conflict of interest

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be thought of as a conflict of interest.

Author contributions

MA: study design and conceptual development. AD: study design and conceptual development. SI: data acquisition and analysis. DK: conceptual development. RG: conceptual development. IH: conceptual development.

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