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Case Report

Central pontine myelinolysis in a chronic alcoholic patient with mild hyponatremia: A case report

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ABSTRACT

Introduction: Central pontine myelinolysis is a type of osmotic demyelination syndrome, which involves damage to parts of brain most commonly pons. The most common causes include rapid correction of hyponatremia but other precipitating factors including alcoholism, diabetes, and chronic liver disease should also be considered.
Case presentation: We present a case of 44-year-old male with a history of chronic alcohol consumption, who presented in emergency room with complaints of slurring of speech and weakness of both upper and lower limbs. His MRI brain reveals 'trident-shaped' appearance with findings of High T2W/FLAIR signal noted in the pons with relative sparing of the periphery and hypo intense on T1W images. He was managed conservatively.
Clinical discussion: Proper diagnosis with MRI is needed for early detection so that proper intervention can be made on time.
Conclusion: CPM can occur in the patient even if they are normonatremic or hyponatremic but can precipitate in Chronic Alcoholic patients.

1. Introduction

Central pontine myelinolysis (CPM) is a rare neurological condition that is characterized by damage to regions of the brain, commonly the pontine white matter, as most frequently caused by the rapid correction of hyponatremia [1]. Osmotic demyelination syndromes are comprised of two disorders namely Central Pontine myelinolysis and extra pontine myelinolysis. Central pontine-type lesions affect the pons and lead to demyelination of structures like the corticospinal tract, corticobulbar tract, and corticopontine tracts at the base of pons [2]. CPM occurs mostly during rapid correction of electrolyte imbalance in particularly susceptible patients like alcoholism, post-liver transplantation, hyperkalemia, diabetes mellitus, chemotherapy, chronic renal failure, hyperemesis gravidarum, and elderly or debilitated people [2,3]. Glial cells especially oligodendrocytes which seems to play a key role in the development of Central pontine myelinolysis are highly susceptible to rapid correction of hyponatremia with hypertonic saline and due to their delayed response to osmotic changes, there is rapid movement of intracellular fluid into extracellular space leading to shrinkage of cells and ultimately apoptosis [4]. Clinical features like dysarthria, dysphagia, flaccid quadriparesis, and spasticity are present during the early course of the disease [2]. The pontine tegmentum is also involved during the late stage of the disease. One of the characteristic presentations of CPM is 'Locked-in syndrome' in which the patient is unable to perform voluntary movements, but the cognitive function and oculomotor function remains intact [1,2]. The chronic alcoholics are susceptible to osmotic stress due to dysfunction of the preventive cerebral mechanism in such patients and due to direct toxicity of alcohol, production of free radicals, and deranged nitric oxide metabolic effects which may favor apoptosis of brain neurons [3]. We present this case to emphasize the importance of diagnosis of CPM in chronic alcoholics.

2. Case presentation

A 44-year-old male patient, with a history of chronic alcohol consumption, presented to the emergency department of our hospital with complaints of slurring of speech and weakness of both upper and lower limbs for the last 10 days. He had been consuming 3.75 units of alcohol

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every day for the last 10 years. He had no significant medical history in the past and he is not under any medications currently.

On general examination, clubbing was demonstrated. The patient was not oriented to time, place and person at the time of admission. On neurological examination, there was dysarthria, left hemiparesis (power of both upper and lower limbs being 3/5), and right-sided plantar extensor response.

As a routine baseline investigation, complete blood count, random blood sugar, liver function test, renal function test and serology were sent. There was a slight increase in Total Leukocyte Count (TLC) 16,200 cells/cumm and mild hyponatremia (Serum Sodium 133 mEq/L), hypoalbuminemia (Serum Albumin 2.98 gm/dl). Rest of the parameters were within normal limits. On the day of admission, with all aseptic precautions, lumbar puncture was done, and cerebrospinal fluid was sent for analysis to rule out the infective etiology. This showed elevated glucose as 76.7 mg/dl (40–70mg/dl) and normal level of protein. Similarly, his vitamin B_{12} and folate levels were in normal range.

On day two of his admission, Magnetic Resonance Imaging (MRI) of the Brain and Cervical Spine were performed with findings of High T2W/FLAIR signal noted in the pons with relative sparing of the periphery, with a 'trident shaped' appearance (Fig. 1).The area appears hypointense on T1W images (Fig. 2) with post contrast showing no significant enhancement. All features in the MRI were suggestive of central pontine myelinolysis which no extra pontine myelinolysis.

In the emergency, fluid restriction was advised as the patient had mild hyponatremia. The patient's serum sodium level was closely monitored thereafter, which was within normal limits. Our report concludes that CPM can develop even when the patient is mild hyponatremic or even eunatremic.

During his stay in the medical ward, the patient was started on antibiotic cephalosporin for the increased TLC count, Pantoprazole, Lorazepam for irrelevant talking and injection Thiamine and methylcobalamine to rule out Wernicke's encephalopathy and Vitamin B_{12} deficiency respectively. The patient was discharged after six days of treatment with follow-up after a month advised in the neurological outpatient department.

3. Discussion

Central pontine myelinolysis was first described by Adams et al., in 1959 as an acute demyelinating disorder in patients with severe hyponatremia in the setting of chronic alcoholism and malnutrition. Since then, various guidelines have been proposed towards correction of sodium which has substantially improved patient morbidity and mortality [5]. It is a rare condition initially believed to affect the pons only, but recent literature suggests extrapontine involvement such as midbrain, thalamus, and cerebellum [6]. CPM is usually caused due to rapid correction of hyponatremia but rarely it can also occur in patients with hyperglycemia and hepatic failure [7]. In our case, the patient had CPM despite rapid correction of Sodium was done.

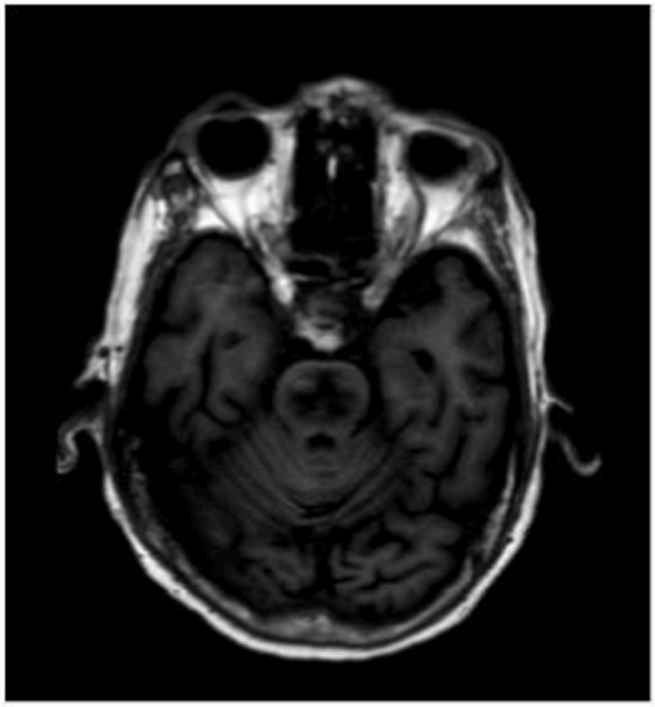

Fig. 2. MRI of the brain showing hypointense T1W signal changes in the pons.

The pathogenesis of developing CPM is not described in the literature. However, the large intracellular shift of water into the brain associated with rapid sodium correction leading to neuronal demyelination is one of the suggested mechanisms for the development of CPM [8]. This leads to the transfer of water from the brain into the cerebrospinal fluid and the shift of intracellular solutes out of cells. In such conditions, if the serum osmolarity comes back to normal very rapidly, as seen in cases of rapid correction of sodium, an osmotic gradient is formed leading to the destruction of the myelin sheath and cell death. The pons, which is the most susceptible structure to the development of CPM, is not efficient enough to transport solutes across membranes leading to localized demyelination of pons sparing other parts of the brain [9].

Chronic alcoholics are more prone to metabolic deficiencies and that likely makes them more susceptible to CPM as this leads to pro-apoptosis of glial cells. They also lack the protective cerebral mechanism to cope with the increase in osmotic stress as they typically present with

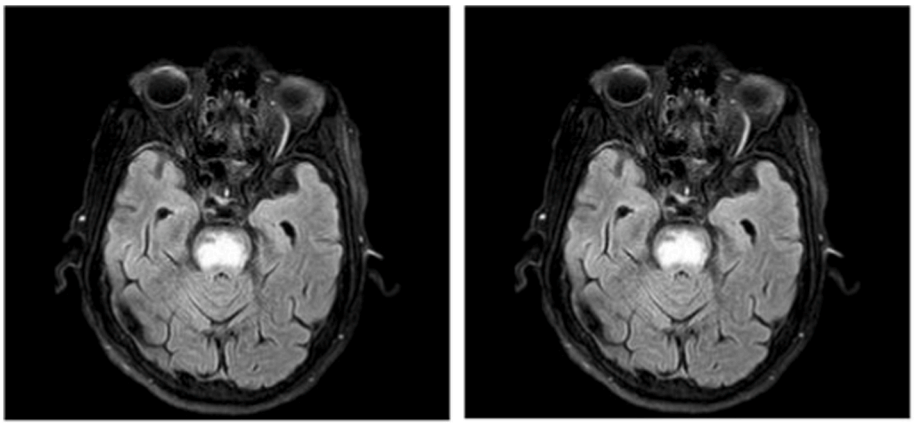

Fig. 1. MRI of the brain showing High T2W/FLAIR signal changes noted in the pons with relative sparing of the periphery in a "trident-shaped" appearance.

malnutrition and have a thiamine deficiency, and are incapable of synthesizing organic osmoles to maintain the osmotic gradient. Moreover, these patients cannot maintain Na+/K + ATPase pump activity due to reduced energy supply. When a chronic alcoholic or malnourished patient develops confusion, quadriplegia, pseudobulbar palsy, or decreased level of consciousness over a period of several days, CPM could be one of the strong differentials [10].

At presentation, our patient had minimal hyponatremia (133 mg/dl) with hypoalbuminemia. Hypoalbuminemia is a common factor in chronic alcoholics which could lead to a hypo-osmolar state, making them more susceptible to CPM. Our patient had raised Total Leukocyte Count but during Lumbar puncture, his CSF revealed normal levels of protein. He had no history of fever with no meningeal signs. Therefore, we excluded the infective etiology. Since our patient was chronic alcoholic, we had a differential of alcohol-induced psychosis. We ruled it out, because our patient didn't present with any hallucinations or agitated behaviour. Wernicke's encephalopathy was our next differential diagnosis. Our patient didn't have any ocular abnormalities, and his symptoms did not improve on administrating Intravenous Thiamine, which excluded Wernicke's encephalopathy.

For the prompt diagnosis of CPM, diffuse weighted MRI is done which shows a characteristic lesion in the pons sparing the ventrolateral pons and the corticospinal tracts within a few hours of the onset of symptoms [11]. MRI changes shows hypointense on T1-weighted images and hyperintense on T2-weighted images in the area of demyelination. In our case, MRI Findings were consistent with CPM and showed High T2W/FLAIR signal in the pons with relative sparing of the periphery, in a 'trident shaped' appearance. The area appeared hypointense on T1W images with post contrast showing no significant enhancement [12–14].

The overall prognosis of CPM is poor. CPM associated with chronic alcoholism has better prognosis than CPM caused due to rapid correction of Hyponatremia [14]. The only recommendation till date is supportive treatment. So, prevention is more focused on identifying people at risk. Sodium correction should be no more than 2–3 mmol/day as recommended by current guidelines [15]. Treatment usually aims at the prevention of complications like deep vein thrombosis and aspiration pneumonia in such patients [16].

There are some limitations of our study worthy to be mentioned. One of the limitation of our study was that we were unable to follow-up the patient due to his family issues and also we couldnot admit the patient for longer duration in the medical ward due to the financial issues from the patients side. With this we could not assess the progression of the disease in the patient.

4. Conclusion

Our case emphasizes that CPM can occur in the patient even if they are normonatremic or hyponatremic but can precipitate in Chronic Alcoholic patients. So, for prompt diagnosis, and different rehabilitation programs which are necessary for the patient, we should focus on MRI rather than focusing on serum sodium concentration which can be normal at times.

Ethical approval

This is a case report, therefore, it did not require ethical approval from ethics committee.

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Author contribution

Author 1: Led data collection, contributed to writing the case information and discussion.

Author 2: Contributed to the process of original draft preparation and introduction.

Author 3: The resident radiologist, who helped in the diagnosis and supervised throughout the process of manuscript writing.

Author 4: Revised it critically for important intellectual content, contributed to review and editing.

Author 5: The resident physician, who help in finding the case edited the rough draft into the final manuscript.

Author 6: Contributed to conceptualization, methodology, and discussion and preserved the pictures.

All the authors read and approved the final manuscript.

Registration of research studies

Not applicable.

Guarantor

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Consent

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the editor-in-chief of this journal on request.

Author agreement statement

We the undersigned declare that this manuscript is original, has not been published before and is not currently being considered for publication elsewhere.

We confirm that the manuscript has been read and approved by all named authors and that there are no other persons who satisfied the criteria for authorship but are not listed. We further confirm that the order of authors listed in the manuscript has been approved by all of us.

We understand that the Corresponding Author is the sole contact for the Editorial process. He/she is responsible for communicating with the other authors about progress, submissions of revisions and final approval of proofs.

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The authors report no conflicts of interest.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.amsu.2022.103736.

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