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Central Pontine Myelinolysis: A Case Report

Harika Karri[1], Bhavya Deepika Cheepurupalli[1], Shaik.Reshma[1], SVBSRN Indraja[1]

[1] *Pharm-D Intern, Department of Pharmacy Practice, Aditya College of Pharmacy, Surampalem, Andhra Pradesh, India*

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ABSTRACT

Demyelination of the central pons is a hallmark of Central Pontine Myelinolysis (CPM), a rare and potentially fatal neurological condition. We report a 60-year-old female patient with CPM, presenting with fever, dysphagia, constipation, and altered sensorium. Laboratory findings revealed electrolyte imbalances, inflammation, and thyroid dysfunction. MRI revealed focal diffusion restriction in the central pons, confirming early CPM. Aggressive treatment with osmotherapy, methylprednisolone, and antibiotics led to significant clinical improvement and normalized laboratory values. This case highlights the importance of multidisciplinary care, timely intervention, and early diagnosis in optimizing prognosis for patients with CPM.

KEYWORDS: Central Pontine Myelinolysis, Osmotic Demyelination Syndrome, Neurological disorder, Electrolyte Imbalance, Multidisciplinary care, Timely intervention.

I. INTRODUCTION

Adams and associates first identified central pontine myelinolysis (CPM) in 1959 as a condition that afflicts malnourished people and alcoholics.[(1)]Central pontine myelinolysis (CPM) is caused by the loss of neurons and myelin that make up the central pontine.[(2)]Central Pontine Myelinolysis (CPM) is a demyelinating condition initially believed to affect only the central pons. It is now also referred to as Osmotic Demyelination Syndrome (OSD) due to the role of severe osmotic stress in its development.[(3)]The most common feature of osmotic demyelination syndrome is focal myelinolysis involving the pons (central pontine myelinolysis); extrapontine myelinolysis can also occur at other sites, like the subcortical white matter.[(4)]CPM symptoms are both neurological, such as confusion, dysarthria, gait instability, and seizures, and neuropsychiatric, including emotional lability and disinhibition. The condition presents with a range of cognitive and behavioural disturbances.[(3)]Chronic alcoholism, malnourishment, hyponatraemia, liver disease, liver transplants, systemic hypotension, and infections are the most prevalent categories of CPM patients.[(5)] Clinical and animal studies support the central role of osmotic stress, most commonly as a result of rapid correction of chronic hyponatraemia, even though the exact mechanism of injury is still unknown.[(3)]

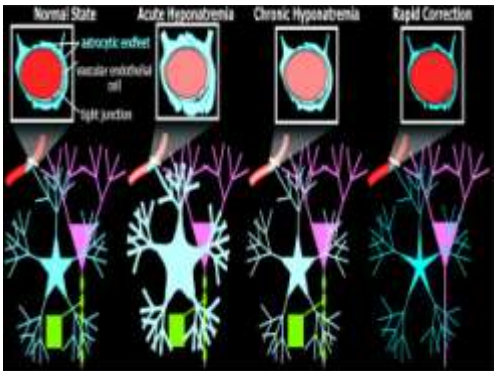

Figure 1 Schematics of major changes that occur in the brain during osmotic regulation

The brain adjusts to variations in blood osmolality during osmotic regulation.Acute hyponatraemia (low blood sodium) causes water to enter the brain, causing astrocyte swelling and cerebral oedema. The brain counteracts this by restoring normal cell volume by losing organic (days) and inorganic (hours) osmolytes. Rapid correction causes water to move out of the brain, which results in shrinkage and dehydration. The brain reabsorbs organic osmolytes slowly and electrolytes rapidly in order to recover. While neurones and axons are spared, rapid correction may cause myelin and oligodendrocyte damage, which would compromise the blood-brain barrier(Figure1).[(3)]

Imaging studies are used to confirm the diagnosis of CPM, MRI is the main diagnostic technique and is better than computed tomography.[(2)]Early diagnosis is greatly aided by magnetic resonance imaging, especially when using

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diffusion weighted images.[6] After the onset of clinical symptoms, standard clinical imaging may remain normal for several days to two weeks. Following this time frame, the typical imaging findings associated with CPM typically surface, with MRI being more likely to be positive than CT.[3]

Patients with severe, potentially fatal hyponatraemia may develop the osmotic demyelination syndrome as a side effect of their treatment. It happens as a result of people with chronic severe hyponatraemia who have made intracellular adjustments to the current hypotonicity, which causes their serum tonicity to rise quickly.[1]

Prevention is key in managing osmotic myelinolysis, making it essential to identify at-risk patients early. Careful intravenous water/electrolyte therapy, with attention to nutritional status, is crucial for reducing risk.

Antioxidant therapy and free-radical scavengers have been proposed, though their effectiveness is debated. Treating the underlying cause of osmotic derangement is crucial, and the approach depends on how long the issue has persisted. Acute osmolar changes can be corrected more rapidly based on the patient's metabolic status.

For asymptomatic hyponatremia, oral correction with a sodium-appropriate diet and fluid restriction is safest for slower correction. A basic metabolic profile should be checked every four hours.

In symptomatic acute hyponatremia, correction should be 1-2 mmol/L/h, not exceeding 8 mmol/L/day. If overcorrection occurs, hypotonic fluids can reduce sodium safely, as rates above 10-15 mmol/L/day are linked to CPM/EPM syndromes.

In acute hypernatremia, sodium levels can be reduced by 1 mmol/L/h, while in chronic cases, reductions up to 10 mmol/L/day or 0.5 mmol/L/h are appropriate. Treatments for hyponatremia include intravenous and oral urea, hypertonic saline, and general fluid restriction.[7]In severe hyponatremia, the correction rate with saline infusion should be adjusted based on symptom severity and the condition's onset speed. This is particularly crucial for patients with risk factors.[6]

II. CASE REPORT

Subjective evidence

A 60-year-old female was admitted to general medicine department with fever and dysphagia to solids for 10 days; constipation and altered sensorium for 3 days; she had a known history of Hypothyroidism since 5years.

Observation

On further evaluation the patient's vital signs were stable.On general examination, there was mild pallor and rest were normal, cardiovascular system and gastrointestinal tract were normal, whereas in respiratory system dyspnoea was found, pt was drowsy but oriented to time, place and person, planter was bilateral flexor, meningeal signs were absent, reflex, power was normal.

On Laboratory examination routine blood investigations complete blood count (Hb-12.3g/dl, TLC-11,200cells/cumm, differential leukocyte count-neutrophils 79%, lymphocyte 18%, platelets 5,46,000lakhs/cumm, ESR-69 mm/hr), liver function test (serum glutamic oxaloacetic transaminase-82 IU/L, serum glutamic-pyruvic transaminase-36 IU/L), and kidney function test (serum creatnine-0.9mg/dl, blood urea-15 mg/dl) ,serum electrolytes (serum Na+=131 meq/l; S.K+=4.5 meq/l),thyroid function test (T_3 – 0.4ng/ml ,T_4 -4.28 µg/dl , TSH-8.94mIU/L),CRP - 151.6mg/l.Viral markers (HIV/HbsAg/Anti-HCV) were negative.

The MRI brain scan revealed a focal area of diffusion restriction in the central pons, sparing the periphery, suggestive of early central pontine myelinolysis (CPM). Differential diagnoses included acute infarct, with demyelination considered less likely. Provisional diagnosis showed Acute infarct in the pons.

Treatment

The patient was admitted and the following treatment was given

Sno	Drug Name	Dose	ROA	Frequency
1	INJ MANNITOL	20%	IV	BID
2	INJ PANTOP	40mg	IV	OD
3	INJ METHYL PREDNISOLONE	500mg in 100ml NS over	IV	OD For 3days

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		30mins		
4	INJ SULBACEF	1.5gm	IV	BID
5	Tab ATORVAS	40mg	PO	OD
6	Tab PCM	650mg	PO	TID
7	Tab TOLVAPTAN	15mg	PO	OD
8	Tab STROCIT	500mg	PO	BID
9	INJ OPTINEURON	1ampule in 100ml NS	IV	OD
10	Syp LACTULOSE	15ml	PO	OD (h/s)

At discharge, the following regimen was prescribed

Sno	Drug Name	Dose	ROA	Frequency
1	Tab SYNDOPA	110mg	PO	BD
2	Tab THYRONORM	25/50mcg	PO	OD alternate days
3	Tab PANTOP	40mg	PO	OD
4	Tab ECOSPORIN AV	75/20mg	PO	OD
5	Tab NEUROBION FORTE	-	PO	OD
6	Tab ULTRACET SEMI	-	PO	BD sos
7	ZYTEE gel LA	-	Topical	TID
8	DIAPRO protein powder	2spoons in glass of milk/ water		TID

A follow up after 1 week was advised to review clinical progress.

III. DISCUSSION

This case report illustrates the diagnosis and successful management of central pontine myelinolysis (CPM), a rare and potentially life-threatening neurological disorder characterized by demyelination of the central pons. A 60-year-old female patient presented with a complex clinical picture, including fever, dysphagia, constipation, and altered sensorium, indicative of severe neurological impairment. The patient's laboratory investigations revealed significant abnormalities, including elevated total leukocyte count, platelets, SGOT, TSH, CRP, and ESR, alongside decreased levels of haemoglobin, serum sodium, T3, and T4. These findings suggested underlying inflammation, electrolyte imbalance, and thyroid dysfunction, known risk factors for CPM.Diagnostic imaging played a crucial role in confirming the diagnosis. The MRI of the brain revealed a focal area of diffusion restriction in the central pons, sparing the periphery, consistent with early central pontine myelinolysis.Prompt and aggressive treatment was initiated, comprising osmotherapy with mannitol to manage cerebral edema, methylprednisolone(500mg IV OD x 3 days, tapered to prednisolone [dose] tabs BD to OD x 3 days) to reduce inflammation, and antibiotics to manage potential infections.Upon discharge, oral medications were prescribed to manage residual symptoms and prevent complications, including syndopa for neurological symptoms, thyronorm for thyroid hormone replacement, pantoprazole for gastrointestinal protection, and ecosprin AV for antiplatelet therapy.A one-week follow-up demonstrated significant clinical improvement, with normalized laboratory values, enhanced sensorium, and improved neurological function.

IV. CONCLUSION

This case report serves as a compelling illustration of the transformative power of multidisciplinary care and timely intervention in managing Central Pontine Myelinolysis (CPM), a rare and potentially life-threatening neurological disorder. The successful outcome achieved in this patient underscores the critical importance of a collaborative approach, where healthcare professionals from diverse specialties converge to

provide comprehensive care. Early diagnosis, facilitated by cutting-edge diagnostic tools, plays a pivotal role in initiating aggressive treatment, thereby preventing catastrophic complications and significantly enhancing patient outcomes. In CPM, where every hour counts, prompt recognition of risk factors, followed by swift and targeted intervention, can mean the difference between irreversible damage and remarkable recovery. As such, early diagnosis and aggressive treatment are the cornerstones of optimal prognosis in CPM patients. By embracing a multidisciplinary care model and prioritizing timely intervention, healthcare providers can substantially improve the quality of life for individuals afflicted with this devastating condition, transforming what could be a dire prognosis into a hopeful and resilient outcome.

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