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Therapeutic Effect of Steroids on Osmotic Demyelination Syndrome due to Hyponatremia in Old Age with Almost Complete Recovery

ABSTRACT

The clinical manifestations of Osmotic Demyelination Syndrome (ODS) are known to develop for 2-6 days after a rapid elevation of the serum sodium level. The symptoms are often irreversible or only partially reversible, and they include dysarthria, dysphagia, tetra paresis, behavioral disturbances, lethargy, confusion, disorientation, and coma. Our patient improved following correction of Sodium within 5 days and was sent home. The patient was readmitted with central pontine myelinolysis manifested as quadriplegia with lower cranial nerve palsy. The patient improved almost completely following methylprednisolone (Steroid) therapy.

Key words: Central pontine myelinolysis, Hyponatremia, Metabolic encephalopathy, Prednisolone therapy

INTRODUCTION

A 77-year-old male presented with metabolic encephalopathy following hyponatremia. Clinically, the patient improved following correction of Sodium within 5 days and was sent home. After 6 days of discharge, the patient was readmitted with central pontine myelinolysis manifested as quadriplegia with lower cranial nerve palsy. The patient improved dramatically following methyl prednisolone therapy to complete recovery.

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CASE HISTORY

A 77-year-old male patient came with two episodes of vomiting, with generalized weakness, and irrelevant talk since morning. There were no h/o fever, cough, headache, chest pain, and breathlessness. The patient is known case of hypertension and dyslipidemia for 15 years. The patient was taking Tab. Telmisartan and hydrochlorothiazide (Telma-H) since many years. Patient is not suffering from any other comorbid conditions. Examination revealed hemodynamically stable, no evidence of dehydration. Central Nervous System examination revealed that the patient was conscious, disoriented and talking irrelevant. Lower cranial nerve was not affected. Motor and sensory system were normal, reflexes and plantar were normal. No neck stiffness. The patient was admitted to intensive care unit for further management. His investigations revealed Hb 13.6 g%, WBC: 9320/mm^3, Platelet: 239 K, Creatine: 0.71 mg%, ECG: Normal, and X-Ray Chest: No e/o Pneumonia or consolidation. His serial serum electrolyte is as follows:

Serum Osmolarity: 230 mosm/L, Urine osmolarity: 305 mosmol/L, Urinary Na: 65mEq/L and Serum Na: 101mEq/L [Table 1] The patient was diagnosed to have hyponatremia due to SIADH and partly due to drug induced (Hydrochlorthiazides). The patient received Inj. Ceftriaxone (Monocef) 1 g IV and Tolvaptan (Natrise) 15 mg twice a day was given. The patient started becoming oriented within 24 h and improved over 5 days and was discharged on 5th day with Na.138 [Table 1] meq/L, K: 4 meq/L. After 6 days of discharged, relative brought patient back with H/O inability to eat, cough (dry) while eating or drinking water and drowsiness. Examination revealed drowsy but arousable, respond to simple command, lower cranial palsy with quadriplegia, and Bilateral small pupils reacting to light with up going plantar response. The patient re-admitted to hospital in intensive care for further treatment. Ryle's tube was inserted and routine blood tests were done Hb:13 g% WBC 7800, Platelet count 430 K, Creatine: 0.9, Na-140, K-4, Cl-101, ECG: Normal, and X-ray chest: Increased Bronchovascular marking. Magnetic resonance imaging (MRI) brain revealed altered signal intensity in the pons with significant increase in size and no post-contrast enhancement. Complete pons involvement with mild extension into bilateral brachium pontis was seen. The patient deteriorated over 24 h and became more drowsy.

Table 1: The changes in serum electrolyte values on consecutive days for case report on therapeutic effect on steroid in osmotic demyelination syndrome due to old age with almost complete recovery

Timings	Na (Meq/L)	K (Meq/l)	Cl (Meq/L)
On admission	101	2.8	68
1st day morning	103	2.9	63
1st day evening	110	3.1	69
2nd day morning	120	3.2	71
2nd day evening	127	3.3	82
3rd day evening	130	3.6	98
4th day morning	131	3.8	101
4th day evening	138	4.0	102

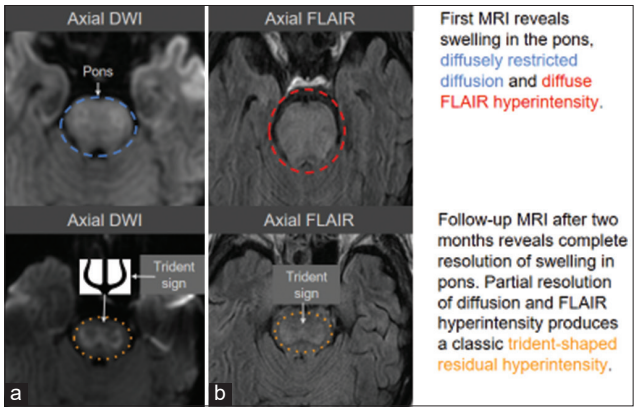

Figure 1: Magnetic resonance imaging reveals swelling in the pons, (a) diffusely restricted diffusion, and (b) diffuse fluid-attenuated inversion recovery hyperintensity

Repeat X-ray chest showed right-sided white out lung suggestive of aspiration pneumonia. The patient intubated and started on higher antibiotics with inotrope with IV fluids. Injection Methylprednisolone was given 40mg IV once daily for 5 days. The patient required tracheostomy for weaning off the ventilator. The patient improved gradually and regain consciousness and quadriparesis improved totally. The patient weaned off the ventilator and weaned off the tracheostomy within 10 days. The subsequent follow-up, MRI brain scan (after 2 months) revealed resolution of the pontine swelling, and partial resolution of the fluid-attenuated inversion recovery (FLAIR) hyperintensity, giving rise to in a classic trident-shaped residual FLAIR hyperintensity. This partial resolution in imaging findings mirrored, but preceded as well as lagged behind the clinical onset and response, respectively.

DISCUSSION

Osmotic demyelination syndrome (ODS) is a rare extensive neurologic deficit. It is the term used for both central pontine myelinolysis and extra pontine myelinolysis.[1] ODS is known to develop following a rapid rise in serum sodium, usually >12 mEq/mL in a 24-h period. Hyponatremia is defined as serum sodium levels below 135 mEq/l.[2] Hyponatremia causes a decrease in extracellular osmolality, causing a shift of fluid from circulation to inside brain cells. A rapid correction of such hyponatremia disturbs the metabolic homeostasis achieved by the brain tissue. The increase in osmolality of circulating blood leads to rapid dehydration of astrocytes and oligodendrocytes.[3]

The pons is considered to be the most sensitive region to this type of injury. The disease is known to have presented 48–72 h after correction of hyponatremia in experiments on rat models.[4] The same presentation was seen in our patient who presented with complaints of paralysis after 6 days of discharge from the hospital. Diagnosis of central pontine myelinolysis is based on clinical suspicion, history of patient, and radiological findings. MRI is the recommended diagnostic modality in suspected patients, earliest changes occurring within 24 h of the onset of quadriplegia in DWI. The regions in T1/T2 axial MRI show a classic [Figure 1] trident-shaped appearance.[5] Clinical features of CPM typically begin to appear within several days after rapid correction of hyponatremia. Clinical manifestations vary and can range from encephalopathy to coma and death. It is postulated therapeutic plasmapheresis could possibly reduce high-molecular myelotoxic substances that are released during osmotic stress.[6,7] The use of glucocorticoids, such as dexamethasone, has also been studied as glucocorticoids are known to impact the permeability of the blood-brain barrier.[8] It is worth giving a trial of Inj. Methylprednisolone for 5 days in this ODS in a desperate attempt to save a life.

CONCLUSION

Osmotic demyelination syndrome (ODS) is a rare clinical entity that involves both pontine and extra-pontine myelinolysis (EPM). Apart from supportive care, there is no proven treatment for established ODS. There is no standard treatment for CPM available at present. It is worth trying steroid therapy to treat ODS. Since Data is limited, it is worth having further prospective studies.

CLINICAL SIGNIFICANCE

Pontine osmotic demyelination is known entity. It is fatal condition which, usually, leads to increase morbidity and mortality. If you start steroid early in the course, then response is better and almost complete recovery can occur.

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