Trident and omega sign: Imaging markers of osmotic demyelination syndrome

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Osmotic demyelination syndrome (ODS) was formerly called as central pontine myelinolysis due to characteristic involvement of pons, however, due to the involvement of both pons and extrapontine structures, it is currently termed as ODS. Extreme osmotic stress due to rapid correction of hyponatremia causes ODS. It can occur in many other disorders such as hemodialysis, correction of hypoglycemia, or hypernatremia and hematological malignancies such as leukemia and lymphoma. ODS is considered primarily a pontine lesion but multifocal involvement is also common. Half of cases have isolated pontine lesions while others have both pontine and extrapontine myelinolytic foci. The extrapontine sites of involvement include basal ganglia and hemispheric white matter thalami, lateral geniculate body, and middle cerebellar peduncle. Here, we report a case of young male who presented with sudden onset of neurological manifestations in emergency department with past history of rapid correction of electrolyte disturbance 1 week back and diagnosed as osmotic demyelination with typical magnetic resonance imaging (MRI) findings.

A 28-year-old male patient presented with complaints of slurring of speech and weakness of bilateral upper and lower limbs for 2 days. He had a history of cellulitis with septic shock 1 week back for which rapid infusion of fluids was given at the local hospital. During the previous admission, his sodium level was 122 mmol/lit, and the rest of the biochemical parameters including the liver function tests, renal function tests were within the normal limits. Rapid correction of sodium with hypertonic saline was done and sodium was normalized to 136 mmol. On examination, vitals were relatively stable with a pulse rate of 72/min and blood pressure of 130/85 mmHg. On neurological examination, a spastic quadriparesis with the cervical sensory defect was noted. Power in the upper and lower limbs was 2/5.

Serum electrolytes, renal function tests, and liver function tests were within normal limits. MRI examination was done which showed T2-weighted imaging (T2WI) and fluid-attenuated inversion recovery (FLAIR) hyperintensity in the central pontine region with sparing of peripheral pons and corticospinal fibers. Axial T2W (Fig. 1a) and FLAIR (Fig. 1b) diffusion (Fig. 1c) images of the brain showing classical trident/omega sign as hyperintensity on T2W and FLAIR and restriction on diffusion images. On basis of the history, clinical findings, and imaging features, a diagnosis of osmotic demyelination/central pontine myelinolysis was made. The patient was managed with supportive treatment with plasma exchange (total of three sessions of fresh frozen plasma exchange of total 1920 ml plasma with 640 ml each session) and immunoglobulins (0.4 g/kg for 4 days) were given and gradually improved in 10 days. The patient was advised physiotherapy.

Extreme osmotic stress most commonly too rapid correction of hyponatremia causes ODS. It is common in middle-aged patients of 30–60 years age group with a moderate male predominance [1]. The most common causes include rapid correction of hyponatremia secondary to alcoholism, liver transplantation, and malnutrition. It is essential to differentiate hypotonic from non-hypotonic hyponatremia. The most common presentation includes altered mental status and seizures, other findings include pseudobulbar palsy, dysarthria, and dysphasia. Basal ganglia involvement causes movement disorders [2]. The outcome of ODS ranges from complete recovery to coma and death. Minimal or no residual deficits are seen in some patients [3].

Imaging features lag 1 or 2 weeks behind clinical symptoms. Non-contrast-enhanced computed tomography shows normal or hypodensity in the affected areas particularly in central pons. On MRI, ODS lesions are hypointense on T1-weighted imaging and hyperintense on T2WI and FLAIR. The lesions are typically well-demarcated and symmetric with pontine involvement gives trident or omega shape as central pontine involvement including transverse pontine fibers with sparing of peripheral pons and corticospinal tracts [4]. Diffusion-weighted imaging shows restricted diffusion in acute cases [5]. In approximately 20% of acute cases, enhancement in the midline and rim of the affected region shows a distinct trident-shaped lesion [6]. Moderate confluent enhancement may be demonstrated in late acute and subacute cases on T1-weighted

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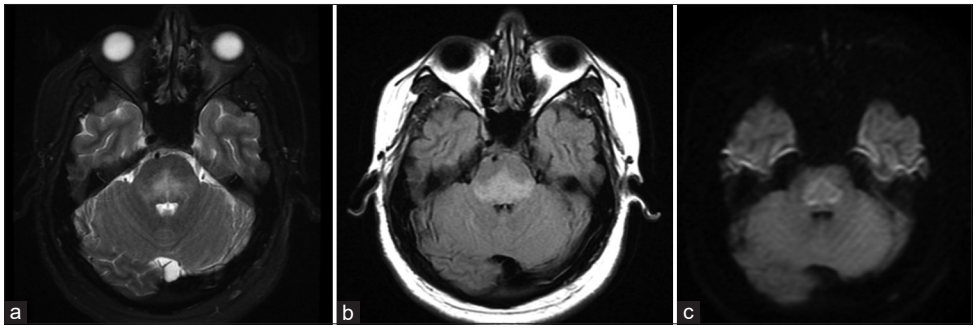

Figure 1: (a) Axial T2-weighted (T2W), (b) fluid-attenuated inversion recovery (FLAIR), (c) diffusion images of the brain showing classical trident/omega sign as hyperintensity on T2W and FLAIR and restriction on diffusion images

contrast-enhanced images. Treatment options include plasma exchange, desmopressin, and immunoglobulins. Supportive treatment with speech therapy and physiotherapy shows prompt improvement [7].

REFERENCES

1. Gankam Kengne F, Soupart A, Pochet R, Brion JP, Decaux G. Re-induction of hyponatremia after rapid overcorrection of hyponatremia reduces mortality in rats. Kidney Int 2009;76:614-21.
2. Chang KY, Lee IH, Kim GJ, Cho K, Park HS. Plasma exchange successfully treats central pontine myelinolysis after acute hypernatremia from intravenous sodium bicarbonate therapy. BMC Nephrol 2014;15:56.
3. Murthy SB, Izadyar S, Dhamne M, Kass JS, Goldsmith CE. Osmotic demyelination syndrome: Variable clinical and radiologic response to intravenous immunoglobulin therapy. Neurol Sci 2013;34:581-4.
4. Miller GM, Baker HL Jr., Okazaki H, Whisnant JP. Central pontine myelinolysis and its imitators: MR findings. Radiology 1988;168:795-802.
5. Chua GC, Sitoh YY, Lim CC, Chua HC, Ng PY. MRI findings in osmotic myelinolysis. Clin Radiol 2002;57:800-6.
6. Ruzek KA, Campeau NG, Miller GM. Early diagnosis of central pontine myelinolysis with diffusion-weighted imaging. AJNR Am J Neuroradiol 2004;25:210-3.
7. Laureno R, Karp BI. Myelinolysis after correction of hyponatremia. Ann Intern Med 1997;126:57-62.

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