A CASE REPORT ON OSMOTIC DEMYELINATION SYNDROME

[1]Bhavani Dhomakonda, [2]Rayeni Srujana, [3]Soumitra Maiti1,
[4]Y. Vidhya Reddy, [5]Tadikonda Rama Rao

[1,2,3,4]Pharm.D., Department of Pharm D, [5]Ph.D., Principal and Professor
CMR College of Pharmacy
Hyderabad, Telangana, India.

Abstract- **A 70 years old man who was admitted to the hospital withknown case of pulmonary tuberculosis since 4 years and COPD since 2 years developed vomiting and hiccups since 8 days patient was apparently asymptomatic 15 days ago later on developed a bilateral pedal edema since 15 days insidious onset gradually progressed up to knee, facial puffiness can be seen, abdominal distension since 10 days insidious onset, urinary incontinence positive, shortness of breath since 8 days insidious onset gradually progressed from grade one and two relieved on taking rest associated with profuse sweating, altered sensorium since 4 days in the of decreased responsiveness not able to walk.. His final diagnosis was osmotic demyelination syndrome (ODS). We discuss the diagnostic and management challenges and the possible complications of this rare diagnosis.**

Keywords: **Osmotic demyelination syndrome, extra pontine myelinolysis, central pontine myelinolysis, systemic lupus erythematosus.**

INTRODUCTION:

Osmotic demyelination syndrome (ODS), which includes extra pontine myelinolysis (EPM) and central pontine myelinolysis (CPM), is a rare neurological condition marked by disruption to the myelin sheath of brain cells brought on by quick changes in plasma osmotic pressure. The primary pathophysiology described is either a decreased neuroglial ability to adapt to significant changes in serum osmolarity or cellular edoema caused by variations in electrolyte forces results in compression and subsequent demyelination of fibre tracts, although it can occur in the presence of a variety of etiological factors. Studies using magnetic resonance imaging (MRI) estimate the incidence to be between 0.3% and 1.1% of hospital admissions. Up to 50% of ODS cases (20-56%) have CPM, while 23-52% of cases have myelinolysis in both locations and pure EPM is less prevalent (13-35% of cases) [1, 2].

CASE REPORT:

A 70 years old male patient with a known case of pulmonary tuberculosis since 4 years and COPD since 2 years developed vomiting and hiccups since 8 days patient was apparently asymptomatic 15 days ago later on developed a bilateral pedal edema since 15 days insidious onset gradually progressed up to knee, facial puffiness can be seen, abdominal distension since 10 days insidious onset, urinary incontinence positive, shortness of breath since 8 days insidious onset gradually progressed from grade one and two relieved on taking rest associated with profuse sweating, altered sensorium since 4 days in the of decreased responsiveness not able to walk. While examination the patient was found to be drowsy, hypertensive (150/70 mmHg), respiratory system positive: bronchial artery embolization, bilateral wheeze positive all the other vitals were found to be stable.

On laboratory investigation, serum creatinine levels were decreased, hyponatremia, hypokalaemia, hypercalcemia was found.

Magnetic resonance imaging (MRI) of the brain showed subtle T2-weighted and fluid-attenuated inversion recovery hyper intensities noted in central pons. Based on the patient clinical presentation and diagnostic methods he was diagnosed with osmotic demyelination syndrome.

His treatment procedure was started with nebulization with levosalbutamol 1.25mg along with ipratropium bromide 0.5mg thrice a day patient was on Ryle's tube feeding with 100ml milk and water and treatment was as follows (Table 1):

Table 1:

DRUGS PRESCRIBED	GENERIC NAME	DOSE	FREQUENCY	ROUTE OF ADMINISTRATION
Syrup. Potchlor	Potassium Chloride	10 ml	TID	Oral
Inj. Furosemide	Furosemide	40 mg	BD	IV
Inj. Ondansetron	Ondansetron	4 mg	BD	IV

Inj. Pantoprazole	Pantoprazole	40 mg	OD	IV
Inj. Augmentin	Amoxicillin/potassium clavulanate	1.2 mg	BD	IV
Inj. MVT	Multivitamin	1 Amp in 100ml Normal Saline	OD	IV
Nebulisation with Duolin, Budecort	Levosalbutamol and Ipratropium, Budesonide	-	SOS	
Inj. Thiamine	Vit.B1	100mg	TID	IV
IVF 2 Normal Saline	Normal Saline	100 ml	OD	IV

DISCUSSION:

Osmotic demyelination syndrome (ODS), which includes extra pontine and central pontine myelinolysis (CPM), is a rather uncommon disorder [3].Osmotic demyelination syndrome patients usually experience encephalopathy or convulsions. Mental status improves as norm natremia is restored and may revert to normal within 48–72 hours, but days later it rapidly deteriorates. Dysarthria, dysphagia, flaccid quadriparesis that progresses to spastic quadriparesis, and horizontal gaze paralysis are symptoms linked to CPM. Coma or delirium usually follow. Tremor, ataxia, and other movement abnormalities such as mutism, parkinsonism, dystonia, and catatonia are characteristics of EPM[4].SLE, hyperemesis gravidarum, anorexia nervosa, Wilson disease, severe burns, hyperphosphatemia and hypokalaemia, diabetes mellitus, renal failure, haemodialysis, and anorexia nervosa are only a few of the various correlations that have been documented[5].It is necessary to correlate radiological evaluation results with clinical findings. The gold standard imaging method for identifying ODS lesions is brain MRI. These comprise lesions that are hyperintense on T2-weighted and FLAIR images and hypointense on T1-weighted imaging. There is no contrast enhancement in the lesions. Each of these characteristics aligns with the results we saw in our instance. Lesions can sometimes emerge during MRI up to two weeks after the commencement of clinical changes. The timing of manifestation can also vary. Radiological results usually do not get better with time, even when the patient fully recovers clinically[6]. Supportive treatment can be provided[7]Individualised care is necessary when treating hyponatraemia. A hypo osmolar patient's neurologic condition, the degree and duration of their hyponatraemia, and other factors are taken into account when deciding on a course of treatment[8].

REFERENCES:

1. Singh N, Kumar A, Baronia AK, Rao PB. Osmotic demyelination syndrome in Intensive Care Unit. Indian Journal of Critical Care Medicine. 2015;19(3):166–9. doi:10.4103/0972-5229.152760.
2. Kalampokini S, Artemiadis A, Zis P, Hadjihannas L, Parpas G, Kyrri A, et al. Osmotic demyelination syndrome improving after immune-modulating treatment: Case report and literature review. Clinical Neurology and Neurosurgery. 2021;208:106811. doi:10.1016/j.clineuro.2021.106811.
3. Lv X, Hong Q, Lin X, Chen W, Tian Y. Osmotic demyelination syndrome: Clinical, neuroimaging characteristics, and outcomes in a series of 18 cases. BioMed Research International. 2021;2021:1–9. doi:10.1155/2021/9944632.
4. Ashraf M, Koul PA, Khan UH, Jan RA, Shah S, Qadri AB, et al. Osmotic demyelination syndrome following slow correction of hyponatremia: Possible role of hypokalemia. Indian Journal of Critical Care Medicine. 2013;17(4):231–3. doi:10.4103/0972-5229.118433.
5. Singh TD, Fugate JE, Rabinstein AA. Central Pontine and extrapontine myelinolysis: A systematic review. European Journal of Neurology. 2014;21(12):1443–50. doi:10.1111/ene.12571.
6. Corona G, Simonetti L, Giuliani C, Sforza A, Peri A. A case of osmotic demyelination syndrome occurred after the correction of severe hyponatraemia in hyperemesis gravidarum. BMC Endocrine Disorders. 2014;14(1). doi:10.1186/1472-6823-14-34.
7. Ngunga M, Harania R, Hameed K, Jowi JO. Osmotic demyelination syndrom: Case report. East African Medical Journal. 2008;84(10). doi:10.4314/eamj.v84i10.9568.
8. Sachan A, Rajitha D, Sunil E, Vengamma B, Devi Bv, Lakshmi A. Isolated extra pontine myelinolysis – a rare imaging appearance of osmotic demyelination syndrome. Journal of Clinical and Scientific Research. 2014;3(1):38. doi:10.4103/2277-5706.241581.