Secondary parkinsonism due to osmotic demyelination syndrome: a case report

Waseem R. Dar, Najeeb U. Sofi, Muzamil Latief, Imtiyaz A. Dar, Moomin Hussain

Department of Medicine, Neurology Division, Shere Kashmir Institute of Medical Sciences, Soura, Jammu and Kashmir, India

Correspondence to Waseem Raja Dar, MD, MACP, Department of Medicine, Neurology Division, Shere Kashmir Institute of Medical Sciences, Soura, Near Police Chowki, 191111 Waterhail Budgam, Jammu and Kashmir, India
E-mail: drwaseem.mw@gmail.com

Received 14 June 2015
Accepted 09 July 2015

The Egyptian Society of Internal Medicine
2015, 27:157–159

Hyponatremia is a common medical problem often found in the elderly and is due to poor intake, medical comorbidities, and medications. Central to the management of this condition is the use of normal and hypertonic saline, besides the use of supplementary salt in diet and limited water intake. However, correction has to be slow; this depends upon whether the patient has acute or chronic hyponatremia. Rapid correction produces a myriad of clinical manifestations, commonly called as osmotic demyelination syndrome. The demyelination is pyramidal in most instances; the basis pontis is usually the frequent location. Extrapyramidal demyelination occurs in 10% of cases. Here, we present a form of extrapyramidal demyelination (i.e. secondary parkinsonism) secondary to osmotic demyelination syndrome, which has rarely been reported in the literature.

Keywords:
hyponatremia, osmotic demyelination, parkinsonism

Egypt J Intern Med 27:157–159

1110-7782

Introduction

Osmotic demyelination syndrome (ODS) is a complication of rapid correction of hyponatremia that occurs as a result of cellular edema, which is caused by fluctuating osmotic forces, resulting in compression of fiber tracts causing demyelination. During the period of hyponatremia, the concentration of intracellular charged protein moieties is altered; reversal cannot parallel a rapid correction of electrolyte status. The central basis pontis is the most common area affected and the resulting clinical syndrome, commonly called central pontine myelinolysis, is characterized by confusion, horizontal gaze paralysis, and spastic quadriplegia. Central pontine myelinolysis is a concentrated, frequently symmetric, noninflammatory demyelination within the central basis pontis, first described by Adams in 1958.

Case report

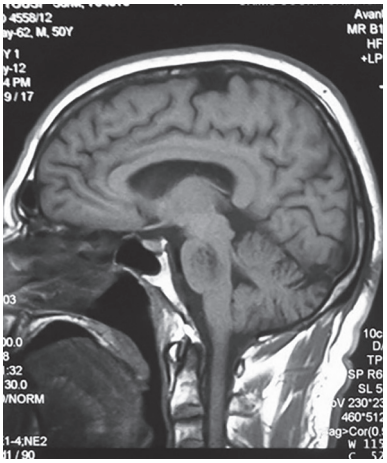
A 50-year-old man presented to our Neurology Department with 3-week history of slowness of all activities, tremors of both hands, and walking with forward posture with small steps. The patient had been on antihypertensive medications (telmisartan+hydro chlorothiazide) for the past 7 years. One month back the patient had been admitted in another hospital with hyponatremic encephalopathy that was corrected with 3% saline. Computed tomography and MRI of the brain were normal at that time. On examination, the patient was conscious and oriented to time, place, and person. There was bradykinesia, bradyphrenia, hypertonia, and bilateral upper extremity tremor. No focal neurological sign was present. Cardiovascular, respiratory, and abdominal examinations were normal. In view of subacute symptoms, secondary parkinsonism was suspected and the patient was evaluated for the same. Baseline investigations were normal (Table 1). Complete blood count showed Hb of 10.7 g%, WBC count of 8200/mm^3, and platelets of 1.89×10^5/mm^3. Metabolic profile revealed the following: sodium, 13 mEq/l; potassium, 3.7 mEq/l; calcium, 9.8 mg/dl; and creatinine, 0.5 mg/dl. MRI of the brain revealed hypointense signals in the pons and the basal ganglia on T1 (Fig. 1) and hyperintense signals on T2 images (Figs 2 and 3) suggestive of demyelination. A diagnosis of parkinsonism secondary to ODS was made and the patient was put on syndopa, pramipexole, and quetiapine. The patient has improved significantly and is currently on our follow-up.

Discussion

Extrapontine myelinolysis (EPM) occurs in 10% of cases of ODS secondary to rapid correction of hyponatremia. Central pontine myelinolysis and EPM are the same disease, sharing the same pathology, associations, and

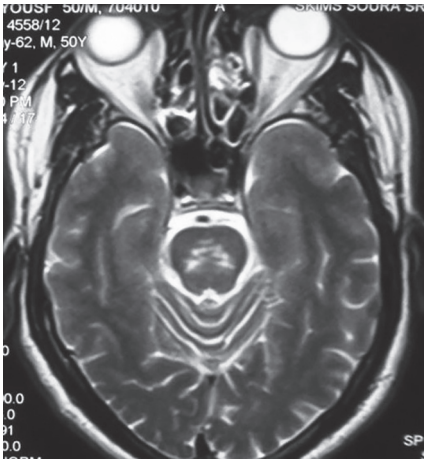
 DOI: 10.4103/1110-7782.174950

Figure 1



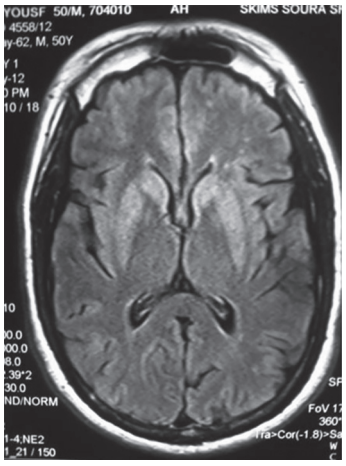
Hypointense lesions in the pons on T1 images.

Figure 2



Bilateral hyperintense lesions in the pons on T2 images.

Figure 3



Bilateral hyperintense lesions in the basal ganglia on T2 images.

time course but differing in clinical manifestations. The mid brain, thalamus, basal nuclei, and cerebellum are the most common regions of the brain involved [1–4].

Table 1 Investigations

Parameter	Result	Parameter	Result	
Hb	10.7 g%	Urea	28 mg/dl	ECG: Sinus Tachycardia
TLC	8200/mm^3	Creatinine	0.5 mg/dl	X-ray Chest: Normal.
DLC	Neutros=68% Lymphos=21% Eosinophils=4%	Bilirubin	1.05 mg/dl	Coagulogram: Normal
PLT	1.89 lacs/mm^3	ALT	40 U/L	USG abdomen: normal
Sodium	133 meq/l	ALP	164 U/L	Urine exam: normal
Potassium	3.7 meq/l	Total Protein	6.76 g/dl	
Calcium	9.8 mg/dl	Albumin	3.8 g/dl	

Mutism, parkinsonism, dystonia, and catatonia have all been reported in case reports as manifestations of EPM [5]. De Souza reported a patient with delayed chorea after recovery from a symmetric parkinsonian syndrome due to striatal myelinolysis following rapid correction of hyponatremia [6]. Emotional and cognitive dysfunctions have also been reported in the literature.

Risk factors for EPM include alcoholism, malnutrition, prolonged diuretic use (as was in this case), immunosuppression, postliver transplant, and posturological surgery [7,8]. It typically occurs between 7 and 14 days after osmotic shift with a characteristic biphasic clinical course. Initially, the patient presents with symptomatic hyponatremia, which improves albeit transiently following sodium correction. This is a followed by a second phase characterized by the features of EPM. During the period of hyponatremia, the concentration of intracellular charged protein moieties is altered; reversal cannot parallel a rapid correction of electrolyte status.

Radiologically, EPM is characterized by hypointense signals on T1 image and hyperintense on T2 image; the lesions are noncontrast enhancing and bilaterally symmetrical [9]. The caudate and putamen are affected with sparing of the globus pallidus. In addition, the corticospinal tract, cerebellum, and subcortical white matter may be affected. The movement disorders of EPM represent a treatable manifestation of the ODS in that a rewarding symptomatic improvement can occur with dopaminergic treatment in those with parkinsonian features. Our patient also improved with symptomatic treatment and is presently doing well.

Conclusion

Hyponatremia is a relatively frequent disorder seen in patients admitted in hospital. Nevertheless, osmotic

demyelination is a relatively infrequent complication of sodium correction. Manifestations of ODS are diverse depending upon the region of the brain involved. EPM occurs less frequently and parkinsonism due to ODS has been scarcely reported in case reports. Prevention is better than cure. Correction of sodium should be slow: 8–10 mEq/l/day in chronic hyponatremia and 10–12 mEq/l/day in acute hyponatremia. Treatment of ODS is symptomatic and prognosis is relatively better in patients with parkinsonism secondary to ODS.

Financial support and sponsorship
Nil.

Conflicts of interest
There are no conflicts of interest.

References

1 West W, Gilbert DT, Wilks RJ. Central myelinolysis in a patient with hyponatraemia. West Indian Med J 2007; 56:382–384.

2 John TM, Jacob CN Xavier J, Kondanath S, Ittycheria CC, Jayaprakash R. A case of acute onset parkinsonism. Qatar Med J 2013; 2013:29-2932.

3 Moulick A, Sengupta P, Banerjee S, Basu D Oromandibular dystonia and persistent psychiatric symptoms in extra-pontine myelinolysis. J Assoc Physicians India 2008; 56:259–261.

4 Ramdurg S, Goyal P, Sharan P. A case of central pontine/extrapontine myelinolysis (osmotic demyelination syndrome) presenting as a catatonic syndrome. Acta Neuropsychiatrica 2011; 23:91–92.

5 De Souza A, Desai PK. Delayed chorea after recovery from a symmetric parkinsonian syndrome due to striatal myelinolysis. J Clin Neurosci 2012; 19:1165–1167.

6 Martin RJ. Central pontine and extrapontine myelinolysis: the osmotic demyelination syndromes. J Neurol Practice 2004; 75:iii22–iii28.

7 Gocht A, Colmant HJ. Central pontine and extrapontine myelinolysis: a report of 58 cases. Clin Neuropathol 1987; 6:262–270.

8 Brown WD. Osmotic demyelination disorders: central pontine and extrapontine myelinolysis. Curr Opin Neurol 2000; 13:691–697.

9 Seiser A, Schwarz S, Aichinger-Steiner MM, Funk G, Schnider P, Brainin M. Parkinsonism and dystonia in central pontine and extrapontine myelinolysis. J Neurol Neurosurg Psychiatry 1998; 65:119–121.