CASE REPORT

Psychogenic polydipsia, hyponatremia and osmotic myelinolysis

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SUMMARY

Objective Description of a case of osmotic myelinolysis associated with hyponatremia produced as a consequence of compulsive water drinking.

Method Case report and review of relevant literature.

Results Compulsive water drinking or psychogenic polydipsia is a common cause of hyponatremia among individuals with chronic mental illness. Central pontine myelinolysis and extrapontine myelinolysis are serious neurological complications resulting from rapid correction of serum sodium and associated changes in serum osmolality. A case of extrapontine myelinolysis confirmed by characteristic MRI findings following an episode of extreme hyponatremia caused by psychogenic polydipsia is described involving a patient with an adult lifelong history of chronic mental illness diagnosed as schizoaffective disorder. With supportive care the related cognitive deficits and balance difficulties resolved completely.

Conclusions Clinicians should be aware of the potential for hyponatremia resulting from compulsive water drinking to cause myelinolysis with delayed development of cognitive and gait symptoms that responds to supportive care if identified early.

the patient's psychiatric status was also improved following the insult and recovery.

CASE PRESENTATION

A 49-year-old man presented to the emergency department of an academic medical centre hospital with altered mental status increasing in severity with a pattern of waxing and waning over the past 2 days. The patient had a longstanding history of schizoaffective disorder and had been managed with valproate 2500 mg nightly and ziprasidone 80 mg twice daily. A report from the patient's family with whom he had been living indicated that the patient had increased his fluid intake to up to 8 L of water daily. The patient also had a history of alcohol use disorder though he had remained free of use of alcohol for several years. This patient had previous presentations of primary psychogenic polydipsia. At this time the patient appeared delirious and was found to have a serum sodium of 101 mEq/L. A head CT scan ruled out intracranial pathology. Admission to the internal medicine service was arranged, to address the hyponatremia. Oral fluid restriction of 1.5 L/day was instituted and normal saline infusions were started. Sodium normalised to 134 within 6 days during which time an agitated delirium persisted requiring soft restraints at times. After correction of electrolytes the patient was transferred to the behavioural health service. On arrival he demonstrated persistent cognitive deficits and episodic agitation. He was observed to have significant gait and balance difficulties and was falling frequently. A brain MRI was obtained and found to be unremarkable. Ziprasidone was discontinued and low-dose haloperidol 1 mg twice daily was begun. Clozapine was initiated at the request of his outpatient provider as he had failed several trials of first and second generation antipsychotics. This was titrated to 350 mg/day. The patient continued to display confusion and increasing instability of gait. Owing to limited improvement and in the light of worsening neurological findings, clozapine was discontinued. The patient demonstrated no return of psychotic symptoms. A repeat brain MRI was obtained which demonstrated new diffuse increased T2 signal enhancement throughout the Lentiform Nucleus. This patchy increased T2 signal in periventricular white matter and right frontal subcortical white matter represented demyelination along with mild diffuse atrophy (figure 1). In the context of clinical correlation this is interpreted presumptively as osmotic demyelination. The patient's subsequent hospital course over the following 10 days was one

BACKGROUND

Compulsive water drinking or psychogenic polydipsia is commonly observed among patients with chronic mental disorders. Estimates of the prevalence of drinking in excess of 3 L a day approach 20% among patients hospitalised for serious psychiatric disorders. Among these individuals a substantial minority develop clinically significant hyponatremia with associated confusion, delirium and ataxia that may progress to seizures, coma and death.¹ Destruction of myelinated structures in the brain may develop in patients who have chronic hyponatremia or have had rapid correction of hyponatremia. This neurological complication has been described as central pontine myelinolysis when affecting midbrain structures and has been termed extrapontine myelinolysis when affecting areas outside the pons, most commonly within the striatum.² We describe a case of extrapontine myelinolysis presenting in an individual with a history of schizoaffective disorder and psychogenic polydipsia resulting in extreme hyponatremia. A syndrome of delirium and gait dysfunction with characteristic enhancement of T2 MRI within extrapyramidal structures is documented. A gradual improvement in cognition and motor function occurred over a period of weeks following the insult. Importantly,

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Figure 1 T-2 mode of MRIs comparing images from before and after osmotic insult.

of progressive improvement in his cognitive state and improvement in his gait. At discharge, the patient had been off all psychotropic medications and was free of psychotic symptoms.

TREATMENT

Treatment was primary supportive in nature while all psychotropic medications were discontinued.

OUTCOME AND FOLLOW-UP

Three months following discharge from the hospital the patient continued to be free of signs or symptoms of gait disturbance. His cognitive examination was normal. He remained free of psychotic or mood symptoms that had troubled him throughout his adult life without need to restart prior antipsychotic pharmacological treatment.

DISCUSSION

First described by Adams, Victor and Mancall, destruction of heavily myelinated brain structures has been repeatedly reported in the medical, neurological and imaging literature among patients who have had rapid correction of hyponatremia. Similar presentations of osmotic demyelination are described in individuals with alcohol use disorders, alcohol withdrawal, malnutrition and diabetic hyperosmolar states.³ Development of central myelinolysis has been associated with aggressive correction of depressed sodium levels that may produce brain oedema and damage related to the osmotic challenges of hyponatremia itself.⁴ A varied constellation of associated signs and symptoms correlating with brain imaging findings may be delayed following stabilisation of electrolyte and osmotic status.⁵ Subsequent course and prognosis has been variable though significant residual neurological deficits routinely follow.

CONCLUSION

A syndrome of confusion, ataxia and delirium occurring in the context of hyponatremia and compulsive water drinking may be followed by a neurological complication characterised by similar symptomatology and characteristic changes in brain imaging affecting structures involved in coordination of motor function. Such a constellation of findings may be underrecognised among patients with serious psychiatric disorders where psychogenic polydipsia is common. The prognosis for affected patients is varied. Improvements in cognitive and motor function have been reported. This individual had complete recovery of cognitive function with no appreciable residual deficits in balance and gait 1 month after discharge. Importantly, at 3-month follow-up the patient has been free of psychotic and affective symptoms that had previously plagued him for his entire life despite discontinuing all psychotropic medications.^{6 7}

RECOMMENDATION

A serum sodium concentration of less than 135 mEq/L is considered hyponatremia. It is the most common electrolyte disorder encountered in clinical practice.⁸ Several factors must be considered when contemplating correction of serum sodium. The presence and severity of symptoms determine the urgency of intervention.⁹ Patients with acute hyponatremia who present with symptoms of headache, nausea, vomiting, seizures, obtundation and central herniation warrant an aggressive approach to correction. When serum sodium levels are less than 115 mEq/L correction with a brief infusion of 3% saline is recommended with a goal to increase the serum sodium by 2–4 mEq/L over 2–4 h. This is followed by a slow correction not to exceed a total of 10 mEq/L over a 24 h period. The serum sodium should not be increased by more than 18 mEq/L within the first 48 h.⁹ Patients with chronic hyponatremia persisting for more than 48 h are less likely to have severe symptoms. However, these individuals are at greater risk for osmotic demyelination syndrome if serum sodium is corrected more rapidly than recommended. Treatment for these individuals begins with a conservative approach of fluid restriction and correction of the pathology responsible for the hyponatremia.

Learning points

- ▶ Psychogenic polydipsia or compulsive water drinking is common among those with chronic mental disorders.
- ▶ Significant hyponatremia or rapid correction of hyponatremia is associated with osmotic damage to myelinated structures in the brain that result in delayed onset of confusion and gait problems that complicate diagnosis in this population.
- ▶ Prognosis for recovery is varied but full recovery of function may occur with supportive treatment.

Contributors TMP is the primary author. He provided overall leadership, teaching and guidance for the contributing residents, and assembled final copy of the paper. CNS discussed the case with radiology and collected background information. PG facilitated discussions with the radiology department in terms of interpreting imaging and provided some of the background information. JAI provided case report and contributed to data gathering.

Competing interests None.

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