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CASE REPORT

Case Report: Severe hyponatremia from psychogenic adipsia

[version 1; peer review: 2 approved]

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Abstract

Hyponatremia is a common emergency room presentation and carries high mortality. We describe a case of a 56-year-old male patient with who presents with refusal to drink water for several weeks leading to the admission. He was diagnosed with psychogenic adipsia and was treated successfully with fluids, mirtazapine and clonazepam.

Keywords

hyponatremia, adipsia, osmolality, altered mental status, osmotic demyelination, paranoia, stroke, infection

Open Peer Review

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Any reports and responses or comments on the article can be found at the end of the article.

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Introduction

Hyponatremia is a common electrolyte abnormality seen in the emergency department and can carry an estimated mortality of 40–60% depending on the degree of severity¹. Psychogenic adipsia is a rare cause of hyponatremia and represents a subgroup where chronic long term management is critical as these patients are likely to relapse. There has been a reported case where hyponatremia has been corrected with hemodialysis using a high sodium dialysate to prevent osmotic demyelination syndrome^{2,3}. One of the mechanisms involved in psychogenic adipsia is the destruction or improper functioning of osmoreceptors in the hypothalamus that controls the thirst mechanism; this may be a result of a congenital malformation or acquired as in the case of stroke, trauma, or infection^{4,5}.

We report a case of hyponatremia with serum sodium as high as 181meq/l and no neurologic manifestations, after patient refused to drink water in the nursing home.

Case presentation

The patient being described is a 56 year old male with cognitive developmental delay and anxiety who is sent from his assisted living facility with hyponatremia on routine labs and documented refusal to drink water. There were no other complaints. He was afebrile and recorded a blood pressure of 99/97mmHg. Physical exam showed a cachectic male with dry mucous membranes. A complete medication list provided by the patient's assisted living facility included famotidine 40 mg daily, docusate 100 mg daily, and a daily multivitamin.

Laboratory analysis showed a plasma sodium concentration of 181 mEq/L, plasma chloride concentration of 138 mEq/L, and plasma potassium concentration of 4.6 mEq/L. Serum osmolality was revealed to be 359 mOsm/kg. The urine sodium level was less than 20 mEq/L and the urine chloride level was also less than 20 mEq/L. Urine osmolality was 1080 mEq/L. The patient was immediately rehydrated with D5 1/2 normal saline solution not to exceed a correction rate of 6–8 mEq/L of sodium per day. The patient continued to refuse most oral intake and denied thirst. A CT scan was obtained without contrast and showed mild microvascular ischemic disease without evidence of intraparenchymal hemorrhage, acute infarct, or hydrocephalus. No hypothalamic infarct or other mass lesion or focal mass effect were seen. Later in the course of his admission he admitted to severe stress from a recent emotional break up. He was started on mirtazapine 7.5 mg daily and clonazepam 0.25 mg twice daily to address his anxiety and that led to an improvement in appetite and regained thirst mechanism. He was stable when discharged back to his assisted living facility. His sodium remained stable within the normal range when discharged back to his assisted living facility and was normal at 6 months post discharge follow up.

Discussion

The above case describes a patient with profound hyponatremia devoid of thirst and remarkably asymptomatic on neurologic exam. There are at least two very interesting phenomenon that can be discussed through this case. One is the presence of an intense emotional response and its effect on the thirst mechanism. Thirst is a very powerful mechanism meant to protect against hyponatremia. Functional MRI studies have demonstrated anterior cingulate gyrus as the core are associated with the consciousness of thirst⁶. The same area is also implicated in a number of psychiatric disorders like schizophrenia depression and autism⁷. While osmoreceptors sense the plasma sodium levels, the consciousness of thirst involves a very different and complex limbic system involvement. A patient with a plasma sodium concentration of 150 mEq/L or more who is alert but not thirsty has, by definition, a hypothalamic lesion affecting the thirst center⁸. Psychiatric illness affecting osmoreceptors of the hypothalamus appears to be very rare and very few cases have been reported; one of them involved a 17-year-old boy with psychosis who displayed an impaired thirst mechanism similar to the patient described above. When the psychosis was treated and began to resolve, the thirst mechanism returned⁹. A detailed psychiatric history should be very useful in preventing recurrences and identifying cases with psychogenic adipsia.

The case also highlights the cerebral adaptation to chronic hyponatremia that results in absent neurologic sequelae. The latter response involves an initial uptake of sodium and potassium, followed by the later accumulation of osmolytes; mainly myo-inositol and the amino acid glutamine. The delayed efflux of these osmolytes as seen when the sodium is corrected too rapidly, is what results in cerebral edema, seizures and coma¹⁰.

In conclusion, psychogenic adipsia represents a rare cause of severe hyponatremia and this case highlights the importance of psychiatric history in patients who present with severe chronic or recurrent hyponatremia.

Consent

Written informed consent for publication of the patient's details was obtained from the patient.

Author contributions

SM wrote the manuscript, performed the literature search. RS and SA conceptualized and were involved in patient care.

Competing interests

No competing interests were disclosed.

Grant information

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Interesting case. It would be more interesting if authors can comment of the role played by mirtazapine in curing the adipsia. One of the side effects of mirtazapine is increased thirst. Mirtazapine increases dopaminergic neurotransmission and dopamine has role in the modulation of thirst. On reviewing some of the case reports which deals with adipsia and psychiatric disorders (depression, schizophrenia), the treatments or intervention (electroconvulsive therapy) used have one thing in common. They all increase dopaminergic activity, supporting the hypothesis that deficiency of dopamine or lack of dopaminergic activity may be playing role in the pathogenesis of adipsia.

Adipsic hyponatremia is uncommon in patients with psychiatric disorders. Hence secondary causes like tumour, histiocytosis, sarcoidosis involving the hypothalamus should be ruled out. An MRI of the brain with or without contrast would be the most appropriate investigation of choice rather than a CT head without contrast.

Patients with adipsic hyponatremias associated with psychiatric disorders will have a normal ADH response and an appropriate increase in urine osmolality.

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Competing Interests: No competing interests were disclosed.

I confirm that I have read this submission and believe that I have an appropriate level of expertise to confirm that it is of an acceptable scientific standard.

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Sarah Manning and co-authors present a case of psychogenic adipsia and hyponatremia. Overall the case report is well written. Although previously described as cited by authors and also additionally reported by others as listed below^{1,2}, this is a known clinical presentation.

This case report will add to literature on this rare clinical presentation.

Urine osmolality units should be changed to mOsm/kg.

Add other published reports about this presentation in discussion.

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