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Case report

Management of geriatric mania complicated by hyponatremia and psychogenic adipsic hypernatremia

Rashmin Achalia, MD [a], Yogesh Lakkas, MD [b], Om Prakash, MD [c,*]

[a] Department of Psychiatry, Government Medical College, Aurangabad, Maharashtra, India
[b] Department of Internal Medicine, Citicare Superspeciality Hospital, Aurangabad, Maharashtra, India
[c] Institute of Human Behaviour and Allied Sciences (IHBAS), New Delhi, India

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ABSTRACT

A 70-year-old male presented with first episode mania and hyponatremia, which were later corrected with an infusion of hypertonic saline. His clinical condition deteriorated because of adipsic hypernatremia associated with hypokalemic periodic paralysis. Hypernatremia correction and potassium supplementation were started but could not be achieved because of manic symptoms as the patient's oral intake was poor. He was restarted on sodium valproate and olanzapine. Over the next few days, the patient's manic symptoms improved significantly and oral intake improved, which eventually led to correction of hypernatremia. Clinicians should evaluate the existence of electrolyte imbalance while dealing with a case of geriatric mania.

1. Introduction

Mania in the elderly is a severe form of an affective disorder with respect to psychotic and cognitive symptoms.[1] Electrolyte imbalance is widely recognized to cause or complicate psychiatric and neurological symptoms and disorders, especially in elderly patients.[2] Electrolyte imbalance and late-onset mania are both common conditions but co-occur rarely. In this report, we describe a case of geriatric mania, the management of which was complicated by hyponatremia, hypernatremia, and hypokalemia.

2. Case report

A 70-year-old male was referred to the psychiatry outpatient department with a 4-week history of irritable mood, increased speech output, decreased need for sleep, and increased energy levels. Food and water intake was inadequate. There was no history of any psychiatric disorder. There was no family history of any medical or psychiatric illness. There was no history of any substance abuse. There was no history of intake of any medications, including Ayurvedic and herbal medicines. There was also no associated history of loss of consciousness, forgetfulness, head injury, or urinary/fecal incontinence. The results of a mental status examination at the time of admission revealed increased speech output, increased psychomotor activity, irritable affect, overfamiliarity, delusion of grandiosity, and poor personal hygiene. The patient had a clear consciousness and orientation to time, place, and person. His minimental status examination revealed a score of 28/30. The possibility of the first manic episode was noted, and the patient was admitted for symptom control. Results of his detailed systemic examination, including neurological examination, were found to be normal. Results of various investigations — including complete blood counts, kidney function tests, liver function tests, blood sugar, thyroid function tests, ELISA (enzyme-linked immunosorbent assay) for HIV, and serum vitamin B_{12} — were within normal limits. Hyponatremia (serum Na level, 115 mEq/L) was documented. For correction of hyponatremia, adequate sodium supplementation was given, resulting in levels of up to 123 mEq/L and 125 mEq/L on Day 2 and Day 3, respectively. After a detailed evaluation and investigations, the diagnosis of manic episode, the first episode was considered and the possibility of delirium was ruled out.

For management of mania, the patient was prescribed sodium valproate (500 mg/day) and oral olanzapine (5 mg/day). The patient developed excessive sedation with psychotropic medications on the 4th day of admission with sodium levels of 131 mEq/L. Hence, an attempt was made to manage his manic symptoms without medications. His sodium levels normalized on the 5th day, i.e.,

* Corresponding author. Department of Psychiatry, Institute of Human Behaviour and Allied Sciences (IHBAS), Dilshad Garden, Delhi 110095, India.
E-mail addresses: drjhirwalop@yahoo.co.in, drjhirwalop@hotmail.com (O. Prakash).

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135 mEq/L. By the 6th day of admission, the patient's manic symptoms were again aggravated in the form of irritable mood, excessive conversation, and refusal to take food. Upon investigation, his serum sodium level was found to be 146 mEq/L and his serum potassium level was 2.9 mEq/L. The patient's serum sodium level continued to rise (150 mEq/L and 155 mEq/L on Day 7 and Day 8, respectively) and his serum potassium levels fluctuated gradually during the subsequent 5 days, with serum sodium 160 mEq/L and potassium 2.8 mEq/L on Day 11. Total leukocyte count (29,400 cmm), hemoglobin level (11 gm %), blood urea nitrogen (90 mg %), and serum creatinine level (2.1 mg %) were also rising. His urine output was found to be 700 mL/day. Serum osmolarity was 354 mmol/kg and urine osmolarity was 860 mmol/kg. Follicle stimulating hormone, luteinizing hormone, thyroid stimulating hormone, and aldosterone levels were within normal limits. Results of X-ray chest examination, urine routine, and ultrasonography of the abdomen were unremarkable. Magnetic resonance imaging brain showed normal hypothalamus and nonspecific periventricular ischemic changes in the cortical regions. A physicians' consultation was sought, and a diagnosis of prerenal azotemia secondary to adipsia was considered.

On the 11th day of admission, the patient developed hypokalemic periodic paralysis, leading to functional intestinal obstruction. Because the patient remained excessively drowsy for the next 2–3 days, psychotropics were again discontinued. A nasogastric feeding tube was inserted, and hypernatremia correction and potassium supplementation was started. Because of active psychopathology, the patient refused to drink water or take food and his irritability also increased significantly. Hence, he was restarted on sodium valproate (tablet, 500 mg/day), and olanzapine (tablet, 5 mg/day) was subsequently added after 3 days. Over the next few days, the patient's manic symptoms improved to some extent and oral intake improved, which further led to correction of hypernatremia and hypokalemia. Over a period of 3 weeks of hospitalization, the patient's irritability was significantly reduced and returned to normal levels in the following 2 weeks. His serum sodium level reached 139 mEq/L and his serum K level was 3.8 mEq/L. He was discharged on sodium valproate and olanzapine. At the 10th month of follow-up, his condition was stable and euthymic.

3. Discussion

Psychiatric symptoms attributable to hyponatremia are relatively common and usually secondary to physical pathology or psychogenic polydipsia.[3] Although there is evidence[4] to support the suggestion that mood changes identical to those seen in bipolar disorder may be caused by hyponatremia at a variety of concentrations. Geriatric mania with hyponatremia complicated by adipsic hypernatremia is poorly documented. The index patient, however, represents such a case.

In the index patient, a manic episode was aggravated by hyponatremia. The plausible explanation for hyponatremia is that because of his manic symptoms, the patient was not taking food and water, which led to dehydration. Because of the severe dehydration, the pituitary gland secreted more antidiuretic hormones as the body's first response to conserve water. Hence, relative salt loss was greater as compared to free water, leading to hyponatremia. Later, hyponatremia was corrected and the patient was put on psychotropic drugs. However, the psychotropic drugs led to excessive sedation, which forced us to stop this treatment. The patient continued to refuse to take anything by mouth and never felt thirsty, which led to adipsic hypernatremia that was proven with laboratory tests. This further complicated the course of the manic episode.

As discussed earlier, the initial response of the body is to secrete antidiuretic hormones when dehydration exists, and the next response is thirst, which is mediated through osmoreceptors situated in the hypothalamus. If thirst is impaired, either due to hypothalamic lesion or drugs affecting hypothalamic osmoreceptors, it will lead to adipsic hypernatremia. In the index patient, hypothalamic lesions were ruled out from the results of the magnetic resonance imaging. There was a possibility that antipsychotic drugs (antidopaminergic) might have suppressed the thirst center, thereby leading to adipsic hypernatremia. However, the patient was maintained well on olanzapine at follow-up and demonstrated normal thirst on continued administration of olanzapine. Hence, without any evidence of underlying physiologic abnormality, the psychogenic cause[5] of adipsia was the most common etiology leading to electrolyte imbalance in this patient. In adipsic hypernatremia, there is a chronic loss of potassium through renal tubules that explains the hypokalemia present in our patient that led to paralytic ileus.

To the best of our knowledge, this appears to be the first case report of geriatric mania presenting with hyponatremia that was further complicated by adipsic hypernatremia. One reported case[6] of adipsic hypernatremia described complication by hyponatremia in a case of tuberculosis meningitis, causing hypothalamic damage. Although late-onset mania usually occurs as a phase of manic–depressive disease, it can occur in association with organic dysfunction-medical and pharmacological-in patients[7] with no history of affective disorders. Clinicians should be wary of older adults presenting with a new diagnosis of mania and should investigate to exclude serious physical pathology.

Conflicts of interest

None.

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References

1. Prakash O, Kumar CN, Shivakumar PT, Bharath S, Varghese M. Clinical presentation of mania compared with depression: data from a geriatric clinic in India. *Int Psychogeriatr* 2009;**21**:764–7.
2. Brooks III JO, Hoblyn JC. Secondary mania in older adults. *Am J Psychiatry* 2005;**162**:2033–8.
3. Illowsky BP, Kirch DG. Polydipsia and hyponatremia in psychiatric patients. *Am J Psychiatry* 1988;**145**:675–83.
4. Rebecca F, McKnight RF, Hampson S. Hyponatremia-induced change in mood mimicking late-onset bipolar disorder. *Gen Hosp Psychiatry* 2011;**33**:83. e5–7.
5. Farley PC, Lau KY, Suba S. Severe hypernatremia in a patient with psychiatric illness. *Arch Intern Med* 1986;**146**:1214–5.
6. Hirsch DJ, Jindal KK. Adipsic hypernatremia complicated by hyponatremia. *Am J Kidney Dis* 1990;**15**:369–71.
7. Siegel AJ. Hyponatremia in psychiatric patients: update on evaluation and management. *Harv Rev Psychiatry* 2008;**16**:13–24.