Auditory Hallucinations Induced by Hyponatremia

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Hyponatremia (serum sodium < 135 mEq/L) is a commonly encountered electrolyte abnormality in day-to-day clinical practice.[1] Studies suggest that around 15%–30% of patients in hospital settings, especially intensive care units, present with hyponatremia.[1] Patients with hyponatremia manifest various symptoms, ranging from mild symptoms like nausea, vomiting, loss of appetite, fatigue, and lethargy to severe symptoms such as confusion, seizures, coma, and death.[1,2] Previous studies[3–7] also reported nonpsychiatric patients with hyponatremia presenting with psychiatric symptoms such as irrelevant talk, poor reality testing, perplexity, incoherent speech, religious and megalomanic delusions, emotional lability, catatonia, panic attacks, and depression. Hallucinations are rarely reported among nonpsychiatric patients with hyponatremia. A previous study[8] reported 0.5% of patients with hyponatremia presenting with hallucinations. We report the case of an elderly woman with no past or family history of psychiatric illness who presented with well-formed auditory hallucinations in the setting of profound hyponatremia.

Case Report

A 72-year-old woman, with a history of hypertension, coronary artery disease, recurrent paroxysmal atrial fibrillation with left-sided pyelonephritis 4 years ago, and hyponatremic encephalopathy 2 years ago, presented to the outpatient psychiatry department with complaints of auditory hallucinations (hearing family members and neighbors talking about her and commanding her) for the past week. She also reported significant tiredness for the last 2 days and recurrent vomiting episodes for the last day. There was no history of headaches, vertigo, fever, head trauma, urinary symptoms, or use of illicit substances. There was no history of psychosis, schizophrenia, mania, depression, anxiety, or insomnia. On examination, she was drowsy, her vital signs were stable, and there were no significant abnormalities in the systemic examination. Considering the abrupt onset of auditory hallucinations and drowsiness, she was transferred to the emergency department (ED) for detailed medical evaluation. While waiting in the ED, she developed an episode of generalized tonic-clonic seizure and was treated with intravenous lorazepam, after which she developed hypotension and required norepinephrine support. Her laboratory investigations revealed severely symptomatic profound hyponatremia (serum sodium: 117 mEq/L, serum osmolality: 257 mOsm/kg, urine osmolality: 268 mOsm/kg, urine sodium: 69 mEq/L, uric acid: 3.9 mg/dL) consistent with syndrome of inappropriate antidiuretic hormone secretion. Her electroencephalogram showed mild diffuse nonspecific cerebral dysfunction.

She was managed as a case of hyponatremic seizure and was given 3% saline with serum sodium monitoring every 4 hours. A proactive relowering regimen with vasopressin was also initiated in view of rapid correction of sodium. Her serum sodium levels gradually improved, and the auditory hallucinations subsided once the serum sodium level normalized. There were no further episodes of vomiting or seizures.

During evaluation for the cause of the patient's hyponatremia, contrast-enhanced computed tomography of the abdomen was taken, which showed a bulky right adrenal gland with calcification, probably a sequela of a healed granulomatous infection. Mantoux test was negative. Laboratory investigations showed serum cortisol: 11 microg/dL, adrenocorticotropic hormone: 18 microg/dL, and dehydroepiandrosterone: 155 microg/dL. As the hormone levels did not indicate significant hypopituitarism, it was planned, in liaison with the endocrinology department, to evaluate the hypothalamic-pituitary-adrenal axis if hyponatremia recurred. Magnetic resonance imaging of the brain showed acute infarcts in the bilateral occipital region, and the patient was started on dual antiplatelet therapy and a statin. She improved symptomatically and was discharged on day 6 of hospitalization.

Discussion

Our patient developed auditory hallucinations acutely in the absence of any past or family history of psychiatric illness that resolved with the correction of hyponatremia. The temporal correlation of onset of hallucinations with hyponatremia and resolution with the normalization of sodium levels without use of antipsychotic medications suggest a causal relationship. Another rare possibility in our patient is that the acute infarcts in the bilateral occipital region precipitated the auditory hallucinations. However, infarcts in the occipital area usually precipitate visual hallucinations, and, to the best of our knowledge, there is only one prior publication that

reported auditory hallucinations in a patient with occipital infarcts.[9]

The mechanism by which hyponatremia causes hallucinations is unclear. However, mutations in genes encoding voltage-gated sodium channels were found to be important in the etiology of psychosis, as it leads to an imbalance of neural excitation and inhibition leading to increased excitability of glutamatergic neurons.[10,11]

In conclusion, there was a clear association between hyponatremia and auditory hallucinations in our case. Clinicians should be aware of such rare presentations of hyponatremia and should evaluate serum sodium levels among patients, especially the elderly, presenting with new-onset auditory hallucinations.

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References

1. Sahay M, Sahay R. Hyponatremia: a practical approach. *Indian J Endocrinol Metab*. 2014;18(6):760–771.
2. Siegel AJ. Hyponatremia in psychiatric patients: update on evaluation and management. *Harv Rev Psychiatry*. 2008;16(1):13–24.
3. Singh RK, Chaudhury S. Hyponatremia-induced psychosis in an industrial setting. *Ind Psychiatry J*. 2009;18(2):137–138.
4. Mauracher L, Rettenbacher M. Case report: manic episode with psychotic symptoms induced by hyponatremia. *Neuropsychiatr*. 2020;34(2):85–87.
5. Peritogiannis V, Rizos DV. Catatonia associated with hyponatremia: case report and brief review of the literature. *Clin Pract Epidemiol Ment Health*. 2021;17(1):26–30.
6. Chen LF, Chi HC, Lin CE. Hyponatremia masquerading as panic symptoms in bipolar affective disorder. *J Neuropsychiatry Clin Neurosci*. 2014;26(4):E3.
7. Sawant NS, Parkar SR, Rupani K, et al. Hyponatremia misdiagnosed as depression. *Ann Indian Psychiatry*. 2019;3(2):168–170.
8. Ellis SJ. Severe hyponatraemia: complications and treatment. *QJM*. 1995;88(12):905–909.
9. Ido F, Badran R, Dmytruk B, et al. Auditory hallucinations as a rare presentation of occipital infarcts. *Case Rep Neurol Med*. 2018;2018:1243605.
10. Imbrici P, Camerino DC, Tricarico D. Major channels involved in neuropsychiatric disorders and therapeutic perspectives. *Front Genet*. 2013;4:76.
11. Suslova M, Hautvast P, Gaebler A, et al. Electrophysiological characterization of schizophrenia-associated variants in NaV1.2 sodium channel. *Eur Psychiatry*. 2022;65(suppl 1):S762.