

## Catatonia Associated with Hyponatremia

Sir,

Catatonia as a clinical entity is associated with affective disorders, psychotic disorders, and organic conditions. Acute organic catatonia is often associated with metabolic, neurological, and toxic conditions.<sup>[1]</sup> Although acute organic catatonia has been linked to various causes, there is a lack of literature on the association of acute organic catatonia with hyponatremia. In this report, we present a case of acute organic catatonia associated with hyponatremia, which improved on correction of the hyponatremia.

### CASE REPORT

A 67-year-old lady presented with signs and symptoms suggestive of Alzheimer dementia for 5 years and was on treatment with Tab. donepezil 10 mg/day for the last 4 years. Since a year prior to the presentation, she was started on Tab. quetiapine 50 mg at night time for sleep disturbances and behavioral problems. She presented to the emergency outpatient services with acute onset symptoms of 1 month duration, characterized by posturing, mutism, staring, negativism, and urinary and fecal incontinence. History revealed a reduction in oral intake over the last 10–15 days. There was no associated history of fever, sore throat, running nose, or symptoms suggestive of urinary tract infection or skin lesions. In terms of psychiatric symptoms/syndromes, there was no history suggestive of depressive symptoms, sudden worsening of cognitive symptoms, or psychotic

symptoms. There was no history of head injury, epilepsy, substance use, hypo- or hyperthyroidism, excessive sweating, or any other medication intake or overdose of medications.

On examination, she exhibited posturing, mutism, waxy flexibility, grasp reflex, and negativism. Further, there was evidence of dehydration and low blood pressure (100/66 mmHg). Physical examination did not reveal any evidence of neck rigidity or gross nutritional deficiencies. Neurological examination was not suggestive of any motor deficit. A provisional diagnosis of organic catatonia was considered. Her Bush Francis Catatonia Rating Scale (BFCRS) score was 21.

On investigation, her hemogram, liver function test, blood glucose levels, X-ray chest PA view, electrocardiogram, and computerized tomography scan of the brain did not reveal any abnormality. However, she was found to have raised serum sodium levels (170 mmol/L), raised serum urea (180 mg/dl), and raised serum creatinine levels (1.7 mg/dl). Other electrolytes were within normal range. Her arterial blood gas analysis also did not reveal any abnormality. Her blood culture reports mentioned “sterile.” There was no evidence of autonomic fluctuation during the initial few hours of assessment.

In terms of management, all medications were stopped. Though lorazepam challenge test was considered,

in view of very high sodium, it was postponed and we decided to carry out the same after correction of serum sodium levels. In liaison with the internist, she was started on normal saline and 5% dextrose every 6 hours. During the first 24 hours, her sodium levels reduced to 164 mmol/L, and by 48 hours, her serum sodium levels reduced to 152 mmol/L. Surprisingly, there was an improvement in posturing, waxy flexibility, and mutism. By the 4<sup>th</sup> day, her serum sodium levels normalized (i.e., reached 142 mmol/L) and all the catatonic features resolved, with a BFCRS score of 1. Further exploration of the history at this time also did not reveal any depressive symptoms, delusions, hallucination, or any other psychotic symptoms. After another day of observation, she was sent home with a final diagnosis of organic catatonia. Quetiapine was started at a dose of 25 mg/day and increased to 50 mg/day after a week. Donepezil was started at 5 mg/day after 5 days of improvement of hypernatremia, and after 3 days, it was increased to 10 mg/day. There was no re-emergence of hypernatremia or catatonia.

## DISCUSSION

Although there are reports of association of catatonia with hyponatremia,<sup>[2-9]</sup> there is limited data on the association of catatonia with hypernatremia. In a PubMed search, we could only find one case report documenting the association of catatonia with hypernatremia (160 mmol/L) in an 87-year-old female. This patient was also diagnosed with dementia and had reduced intake with a background of fever. In terms of catatonic features, the patient had posturing, psychological pillow, mutism, waxy flexibility, staring grasp reflex, and advertence reaction.<sup>[10]</sup> The case presented by us also had poor oral intake and showed a similar picture of catatonia. The previously reported case in the literature was also managed with normal saline and dextrose, as in our case. In our patient, all symptoms of catatonia resolved over 3 days. The previous report also suggested a similar picture.<sup>[10]</sup>

Hypernatremia is defined as an increase in the serum sodium levels above 145 mmol/L. It is considered to be a hyperosmolar condition caused by a reduction in the total body water, relative to the electrolyte content of the body. Accordingly, hypernatremia is considered as a state of reduced body water, rather than a deficiency of sodium homeostasis. It is usually seen in elderly patients who have physical or mental illnesses, with associated impaired thirst with or without restricted access to water. At times, fluid loss due to any cause can exacerbate the hypernatremia.<sup>[11]</sup>

In the index case, the patient had symptoms of 1-month duration, which started with catatonic features which

were possibly preceded or associated with decreased oral intake. Although such history was not available, it is possible that both the catatonia and hypernatremia were an outcome of dehydration as a result of poor intake. Accordingly, correction of dehydration with saline and dextrose led to a correction of sodium as well as improvement in the catatonia. However, it is also possible that catatonia was associated with hypernatremia, and the presence of catatonia could have led to dehydration due to poor intake. In the index case, catatonia responded to correction of hypernatremia, which provides further evidence for the association of catatonia with hypernatremia.

As hypernatremia reflects hyperosmolarity, it leads to shrinkage of neurons and resultant brain injury. Risk factors for the development of hypernatremia include advanced age, mental and physical impairment, presence of uncontrolled diabetes mellitus, polyuria due to any cause, use of diuretics, and hospitalization or admission to a nursing home where the patient is provided with inadequate nursing care. Hypernatremia is considered to have a poor prognosis when it is associated with low systolic blood pressure, low serum pH, very high serum sodium levels (>166 mmol/L), high plasma osmolality, dehydration, or pneumonia.<sup>[11]</sup> The clinical features of hypernatremia can be broadly divided into features of dehydration (such as tachycardia, hypotension, dry mouth, and low skin turgor) and cognitive and neurological symptoms (such as confusion, obtundation, lethargy, abnormalities of speech, irritability, myoclonic jerks, seizures, and nystagmus), which could be attributed to the shrinkage of neurons due to dehydration. Some patients may have additional symptoms of generalized weakness and weight loss.<sup>[11]</sup> Our patient had features of dehydration along with features suggestive of pre-renal azotemia, which was possibly related to reduced oral intake.

Management of hypernatremia depends on the rapidity of its development. Acute hypernatremia is defined as developing over less than 24 hours, and chronic hypernatremia is defined as developing over more than 24 hours. Chronic hypernatremia must be corrected slowly, i.e., at a rate of about 8–10 mEq/d.<sup>[11]</sup> In our case, we presumed that the patient has chronic hypernatremia, and accordingly, corrected her sodium at a slower pace.

The index case demonstrates that catatonia in an elderly person may be associated with hypernatremia. While evaluating an elderly person with catatonia, hypernatremia should always be considered as a possible cause, and serum electrolytes, especially serum sodium levels, must be evaluated.

**Declaration of patient consent**

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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**Conflicts of interest**

There are no conflicts of interest.

**Aseem Mehra, Sandeep Grover**

Department of Psychiatry, Postgraduate Institute of Medical Education and Research, Chandigarh, India


**Address for correspondence:** Dr. Sandeep Grover  
 Department of Psychiatry, Postgraduate Institute of Medical Education and Research, Chandigarh - 160 012, India.  
 E-mail: drsandeepg2002@yahoo.com

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