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LETTER TO THE EDITOR

Aphasic syndrome associated with severe hypernatremia secondary to lithium treatment☆

Síndrome afásico asociado a hipernatremia grave secundario a tratamiento con litio

To the Editor:

We have read the scientific letter ''Severe hypernatraemia associated with lithium treatment''[1] with interest, given that its publication coincides with the admission of a patient with similar characteristics to our hospital. The motive for this letter is to report our case due to its atypical clinical symptoms. The patient was an 84-year-old woman with bipolar disorder, diagnosed 30 years ago and treated with lithium for the past 15 years (current dose, 600 mg/day). She was referred to the emergency department for suddenly appearing language disorder. Relevant in the patient's history were high blood pressure treated with amlodipine (10 mg/day), dyslipidemia under treatment with ezetimibe (10 mg/day) and chronic renal failure (CRF). From the psychiatric viewpoint, the patient had been stable for the previous 2 years, with only a 2-week period in the last year in which the dose of lithium carbonate was reduced to 300 mg a day, the periodic dose falling within the therapeutic range. The patient had been diagnosed with nephrogenic diabetes insipidus (NDI) in the context of compensated CRF 2 years earlier, during a hospital admission for severe hypernatremia that required diuretic treatment (furosemide 40 mg/day), which was suspended after 2 months. Characteristic symptoms of the condition, such as polydipsia and polyuria, have persisted from that episode until now. The biochemical analyses performed in the emergency department revealed hypernatremia (sodium [Na^+], 158 mmol/l), chloremia (112 mmol/l) and hyperkalemia (4 mmol/l), with plasma osmolality of 308 mOsm/kg, urinary osmolality deficit (290 mOsm/kg), urinary Na^+ of 24 mmol/l and urine potassium of 20 mmol/l, uremia of 61 mg/dl and creatininemia (1.84 mg/dl). The rest of the values were within normal range.

Patient lithemia was then 0.95 mmol/l. Neurological examination revealed an alert patient, lucid (exam limited due to speech disorder) and attentive, with significant reduction of spontaneous language including phonological paraphasias, deficit in naming object (anomia), altered repetition and disordered comprehension of complex orders. No signs of focal motor or sense deficits were observed; however, cogwheel rigidity was found in all 4 limbs and postural tremor was exhibited in both hands. The rest of the exam results were normal.

The initial diagnosis was expressive aphasia, presumably involving a stroke. Brain nuclear magnetic resonance imaging (NMRI) with a diffusion-weighted sequence was performed, which eliminated acute injury. Given the previously described patient history, a desmopressin test was performed. As the plasma osmolarity remained the same, the diagnosis of NDI secondary to lithium treatment was confirmed; lithium was immediately suspended. We consequently instituted treatment with amiloride 10 mg/day and indometacin 150 mg/day. Osmolarity and electrolyte values returned to normal in 48 h, with complete reversion of the language symptoms. Lithium was suspended, the patient was released with amiloride as treatment for NDI and dose titration of quetiapine was initiated.

This case reaffirms what was described in the letter from Prieto Tenreiro with respect to the hydro-electrolytic imbalance and the response to treatment.[1] We feel that the current decompensation was secondary to the reduction in liquid intake that occurred in the week prior to hospital admission for unknown reasons (she usually drank 4 l a day).

The deficit in fluid supply, in the context of a NDI, increased plasma osmolarity. The mechanisms of brain compensation and the accumulation of intracellular solutes were insufficient to compensate the lack of free fluid. This generated dehydration and cellular dysfunction. The clinical symptoms simulating aphasia has not been described and it seems useful to bear in mind. The physiopathology of this condition is unclear. However, given that the mechanism involved in encephalopathy from hyperosmolarity is the overall passage of water from inside the cell to the interstitium at the brain level,[2] we feel that the phenomenon was produced in this case in a more local manner, affecting sensitive language-related areas.

The sudden presentation of the aphasic symptoms in our patient, without signs of confusion syndrome, made it necessary to eliminate various etiologies (such as vascular, epileptic, migraine and psychiatric), determining an entity known in reports published in English as ''stroke mimic''.

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The importance of being able to detect this condition lies in that in different opportunities patients receive treatments aimed at other etiologies that may be confused with this condition. Clinical assessment, brain MRI, biochemical analysis and treatment response were sufficient for eliminating the possible diagnoses mentioned.

The especial vulnerability of the limbic region[3] to the phenomenon of hypernatremia has previously been described and, based on this case, we suggest studying it for other locations such as the language-related areas.

References

1. Prieto Tenreiro AM. Hipernatremia grave en relación con un tratamiento con litio. Rev Psiquiatr Salud Ment (Barc). 2012;5:254–5.
2. Oh MS, Carroll HJ. Disorders of sodium metabolism: hypernatremia and hyponatremia. Crit Care Med. 1992;20:94–103.
3. Cagnin A, Manara R, Piron L, Dam M. Hypernatremia-induced limbic system damage. Anesthesiology. 2011;114:175.

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