The pathophysiology of this clinical picture is probably associated with the loss of cerebrovascular autoregulation, causing hyperperfusion and leading to rupture of the blood-brain barrier with ingress of fluid and blood breakdown products to the brain parenchyma.[15] Focal cerebral vasoconstriction mediated by brain ischaemia is improbable due to the extension of the oedema and the absence of signs of ischaemia. Endothelial dysfunction has been reported in cases associated with preeclampsia, use of cytotoxic drugs, and autoimmune disease.[1,2]

This clinical case is unusual in that MCI progressed to AD after correction of AHT in the context of pronounced RPLS, in a patient with *APOE* genotype $\varepsilon 4/\varepsilon 4$. With this case report, we aim to raise clinicians' awareness of the early identification of this syndrome, early treatment to avoid neurological sequelae, and the necessary close monitoring of all patients with RPLS through a timely cognitive/neuropsychological assessment.[14] Based on the supposed reversibility of RPLS, there is a risk of underestimating a cognitive disorder and failing to adjust treatment.

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19 October 2017

https://doi.org/10.1016/j.nrleng.2018.01.007
2173-5808/

Guillain-Barré syndrome and hyponatraemia☆

Síndrome de Guillain-Barré e hiponatremia

Dear Editor,

Hyponatraemia, the most prevalent electrolyte imbalance in clinical practice, is associated with increased morbidity and mortality. The association between hyponatraemia and Guillain-Barré syndrome (GBS) has been described in the literature, although few studies have analysed its prevalence, aetiology, pathophysiology, diagnosis, and treatment. We present the case of a patient with GBS and severe hyponatraemia.

The patient was a 59-year-old man with type 2 diabetes mellitus and good metabolic control. He attended our hospital due to paraesthesia in the hands and feet and difficulty walking (2 falls at home), loss of sphincter control (requiring a urinary catheter), general discomfort, and drowsiness. Three weeks previously, the patient had presented diarrhoea, which lasted 3 days and resolved spontaneously. The examination revealed symmetric paraparesis, hypoaesthesia in both hands, and generalised areflexia. The patient was adequately hydrated and did not present oedema. A blood analysis revealed a low sodium concentration at

☆ Please cite this article as: Ternero Vega JE, León RG, Delgado DA, Baturone MO. Síndrome de Guillain-Barré e hiponatremia. Neurología. 2020;35:282—284.

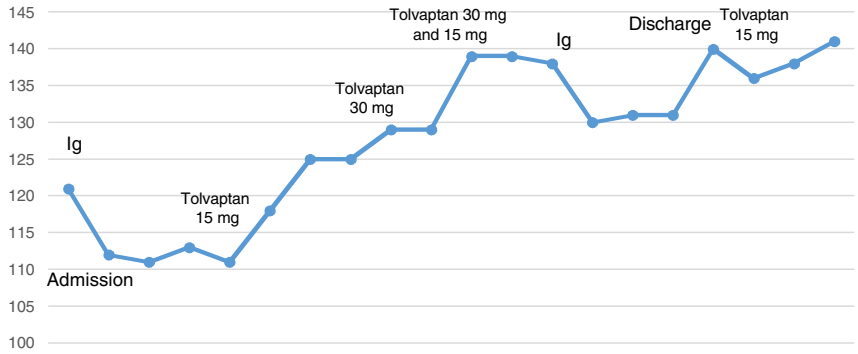

Figure 1 Changes in our patient's sodium levels during hospitalisation.

121 mEq/L (normal range, 135-145 mEq/L) and a glucose level of 168 mg/dL (normal range, 80-120 mg/dL), with normal potassium, urea, creatinine, and total protein concentrations; a head CT scan revealed no abnormalities. A CSF analysis detected albuminocytologic dissociation, a protein level of 2.34 g/L (normal range, 0.15-0.5 g/L), and a cell count of 8 cells/mm^3. The microbiological analysis yielded negative results. Based on these findings, the patient was diagnosed with acute inflammatory demyelinating polyradiculoneuropathy (a form of GBS) and euvolaemic hyponatraemia.

We started treatment with immunoglobulins (Ig) dosed at 0.4 g/kg/day, in 5 boluses. However, treatment only achieved partial motor improvements, and an additional cycle was necessary 2 weeks later. Electroneurography and electromyography studies performed during hospitalisation confirmed the diagnosis. Neurological symptoms improved progressively; at discharge (a month after admission), however, the patient continued to display lower limb weakness, which prevented him from walking.

Physical examination results were compatible with euvolaemic hyponatraemia. Plasma osmolality was 239 mOsm/kg (normal range, 275-295 mOsm/kg), urine osmolality was 591 mOsm/kg (normal range, 100-700 mOsm/kg), urine sodium concentration was 80 mEq/L (normal range, 20-200 mEq/L), and TSH concentration was 1.70 mIU/mL (normal range, 0.4-4mIU/mL); the patient showed a normal lipid profile and no monoclonal proteins in protein electrophoresis. Chest radiography revealed mediastinal widening; a chest and abdomen CT scan revealed no signs of tumour. The patient was diagnosed with euvolaemic hyponatraemia associated with syndrome of inappropriate antidiuretic hormone secretion (SIADH) secondary to GBS. The patient was initially treated with 3% hypertonic saline (500 cc/24 h) and water restriction (800 cc/24 h); we subsequently administered tolvaptan dosed at 15 mg/day due to lack of response. The drug was up-titrated to 30 mg/day, and progressively withdrawn on an outpatient basis until complete discontinuation 4 months later.

One year later, lower limb weakness had improved with rehabilitation therapy, although the patient needed crutches to walk. Serum sodium levels were normal (141 mEq/L).

Although some studies report an association between hyponatraemia and GBS, few studies have analysed the correlation. This association results in poorer hospital outcomes even at one year,[1–5] longer hospital stays,[1–3] and higher costs.[1] The association between hyponatraemia and GBS is even reported to be an independent predictor of mortality,[2,5] which has also been described in other disorders.[1] Some studies suggest that patients with GBS and hyponatraemia are more likely to require ventilatory support,[1,2] which may be explained by the fact that severe hyponatraemia can manifest as respiratory distress.[6]

Most cases of hyponatraemia in the context of GBS develop during hospitalisation and are associated with Ig treatment; pseudohyponatraemia linked to increased protein levels may therefore play an important role. Another possibility is associated with water transport from the intracellular space to the intravascular space due to increased osmolality secondary to infusion of sugar-stabilised Ig. Palevsky et al.[7] analysed the effect of Ig infusion on sodium levels, measured with direct potentiometry to avoid diagnosis of pseudohyponatraemia. The authors observed hyponatraemia, despite using this technique. SIADH is a frequent cause of true hyponatraemia in patients with GBS.[2,8,9] Other cases are due to cerebral salt-wasting syndrome, although this association is very rare.

Our patient may meet the diagnostic criteria for SIADH as established in the latest European hyponatraemia guidelines (plasma osmolality < 275 mOsm/kg; urine osmolality > 100 mOsm/kg; euvolaemia; and absence of adrenal insufficiency, hypothyroidism, hypopituitarism, or kidney failure).[7] We did not determine cortisol levels in our patient; however, clinical and laboratory results were not compatible with adrenal insufficiency. The prevalence of SIADH in patients with GBS is not clear; case reports constitute the only available evidence. The pathophysiological mechanisms of the association between GBS and hyponatraemia are yet to be understood. Several hypotheses have been proposed: alterations of hypothalamic cells, causing ADH release into the bloodstream; alterations in osmoregulation; increased sensitivity of ADH receptors; and other mechanisms not related to ADH.[9] Other researchers support the involvement of interleukin 6, which may increase vasopressin release.[10]

Interestingly, our patient's sodium levels decreased following Ig administration (Fig. 1). This may indicate pseudohyponatraemia and/or water transport to the intravascular space, which play a role in true hyponatraemia. In retrospect, this may explain the patient's poor response to hypertonic saline and water restriction and the

need to up-titrate tolvaptan to 30 mg/day to achieve normal sodium levels.

The available evidence on treatment with tolvaptan in patients with GBS and SIADH is strikingly scarce: to our knowledge, only one case has been published to date.

Sodium levels should be monitored in patients with GBS. Pseudohyponatraemia, water transport, and SIADH should be considered in the differential diagnosis of hyponatraemia. In our case, hyponatraemia may have played a role in the need for an additional cycle of Ig and the slow motor recovery.

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https://doi.org/10.1016/j.nrleng.2017.12.011
2173-5808/

Echocardiographic parameters of atrial cardiopathy and the detection of atrial fibrillation in patients with cryptogenic stroke☆

Parámetros ecocardiográficos de cardiopatía auricular y detección de fibrilación auricular en el ictus criptogénico

Dear Editor,

Cardioembolic stroke accounts for 20%-30% of all ischaemic strokes.[1] With the introduction of the term ''embolic stroke of undetermined source'' (ESUS), and given the considerable number of non-lacunar cryptogenic strokes, the hypothesis of an unknown embolic source has led to extensive study. This includes prolonged cardiac monitoring, which increases sensitivity for identifying paroxysmal atrial fibrillation (AF).[2] Although the ability of these paroxysmal episodes to cause ischaemic events was previously unknown, studies into the pathophysiology of cardioembolic stroke have shown that paroxysmal AF, which usually lasts 5-6 minutes, increases the risk of stroke.[3,4]

Given their short duration and the fact that they are often asymptomatic and may not co-occur with neurological symptoms, these episodes are classified under stroke of undetermined cause.[3,5] The heterogeneity of this group, combined with the limited use of prolonged cardiac monitoring (whether due to patient intolerance or to the unavailability of monitoring equipment and differences between devices), has led researchers to investigate parameters correlated with greater incidence of AF,[6] with a view to increasing the utility of the technique. These parameters include electrocardiographic, biochemical, and echocardiographic variables, which have been found to be associated not only with AF but also with ischaemic stroke and recurrent stroke.[1,7]

We present the case of a patient with acute ischaemic stroke and morphological and functional signs of left atrial cardiopathy in an echocardiography study; these findings were correlated with paroxysmal AF.

The patient was a functionally independent 77-year-old woman who came to the emergency department due to right hemiparesis and mixed aphasia. She scored 1 on the NIHSS due to language impairment, which resolved com-

☆ Please cite this article as: Lambea Gil Á, Tejada Meza H, López Perales CR, Artal Roy J, Marta Moreno J. Parámetros ecocardiográficos de cardiopatía auricular y detección de fibrilación auricular en el ictus criptogénico. Neurología. 2020;35:284—287.