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A Case of Hyponatremia as the Presenting Symptom of Guillain-Barré Syndrome

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Abstract

Guillain-Barré (GBS) syndrome is an immune-mediated polyneuropathy that frequently presents with progressive muscle weakness. Hyponatremia is rarely the presenting feature of this demyelinating process, as it is normally described following the diagnosis. We report a case of Guillain-Barré syndrome diagnosed after presenting symptoms of severe hyponatremia. Awareness should be raised for consideration of both Guillain-Barré syndrome and its treatment with intravenous immune globulin therapy as the cause of hyponatremia.

Keywords

Guillain-Barré syndrome; hyponatremia; inappropriate ADH syndrome (SIADH)

Case Report

A 79 year old female patient visited the emergency department due to generalized weakness, especially intense in the lower limbs, which occurred three days before admission. She mentioned symptoms of upper respiratory system infection a few weeks earlier without diarrheas. Her past medical history included hypertension (for which she wasn't on any therapy), diabetes mellitus, dyslipidemia and carriage of thalassemia and was thus on medications including simvastatin, metformin and folic acid. No diuretic use was mentioned. The clinical examination showed a weakness of 4/5 in lower limbs with normal reflexes, no cranial nerve impairment, negative Babinski sign, GCS 15/15, absence of fever, normal pulmonic, cardiac and proctorectal examination and no clinical evidence of dehydration. There was no opacity attributed to a past pneumonia in her chest X-ray. Concerning her laboratory tests, total cholesterol, CRP, urea, creatinine, γ-GT, ALP, ALT, AST, thyroid function, 8 AM cortisol, parathormone, serum protein electrophoresis, B12, folic acid levels, WBCs and PLTs were within the normal ranges. On the other hand there were abnormal values for CPK: 701 U/L, CK-MB: 56U/L, LDH: 625 U/L, Glu: 246 mg/dl, Na: 119 mmol/l, K: 5,2 mmol/l, ESR: 70, Hb: 9,5 g/dl,WBCs: 6300 (64% neutrophils), urine potassium:119mmol/l (normal ranges 25–125 mmol/l), urine osmolality: 541 mOsm/kg (normal value 50-1400mOsm/kg) ,serum osmolarity: 257 mOsm/kg (normal value 270-295mOsm/kg), osmolarity gap: -28 mOsm/kg. The tests of urine antigens for Legionella and Streptococcus pneumoniae were negative. The cardiac monitoring became abnormal with a troponin test in the grey zone that decreased in 6 hours and a left branch block with normal cardiac echocardiogram, however episodes of flattering in cardiac enzymes followed by ST inversion were observed. Her chest and abdomen X-ray had no obvious

pathology. Initial treatment with 3lt NS 0, 9% /8 hours for two days was initiated, but had no effect on sodium levels. At the same time she had deterioration of her symptoms with episodes of fainting and abdominal pain. Investigation with normal abdominal ultrasound and computed tomography (CT) imaging was normal.

On the 5th day of admission the patient deteriorated with 3/5 muscle strength in the lower and 4/5 in the upper limbs, difficulty in head support, sensory impairment in extremities without any sensory level (pathologic pinprick test, light touch, vibration), decreased tendon reflexes +1 in the lower limbs and constipation. CT of the brain was normal and a lumbar puncture (LP) showed: 0 cells, 111 protein g/l, normal glucose, negative culture and Gram stain and PCR for CMV, EBV and HSV. These findings were consistent of GBS and, immediately after the consultation of a neurologist, immunoglobulin IV 400mg/kg/d for 5 days in combination with water restriction was initiated.

A couple of days after the completion of immunoglobulin therapy, the patient was significantly improved with better sensory ability on the right side, muscle strength 4/5 on the right lower limb and upper limbs and 3/5 on the left lower limb, +2 lower limb tendon reflexes, normal head support and GCS15/15. In the meantime, her potassium rates reached normal levels and both CPK and CKMB became normal. The autoimmune antibodies, especially the anti-Jo for myositis, along with the viral, Mycoplasma and Yersinia serology were non-reactive. The patient was discharged on the seventeenth day with muscle strength 4/5 in lower limbs and 5/5 in upper limbs and cervical muscles, normal reflexes and sensation and no abdominal pain.

The findings of the MRI and the electromyography (EMG) in 2 weeks showed no specific pathology. That can occur in 1% of patients, especially if they have significantly milder weakness at nadir to patients with an abnormal nerve conduction study [1].

Discussion

Hyponatremia in patients diagnosed with GBS is well described in one prospective study. Precisely, hyponatremia was noted in 48% of cases [3] concerning 50 patients diagnosed with GBS, while motor dysfunction preceded the onset of hyponatremia, and only seventeen cases of GBS-related SIADH have been published since 1964. Only two prior case reports of hyponatremia with neuromascular deficits as manifestation were published.

The possible underlying etiology of this phenomenon is thought to be the syndrome of inappropriate secretion of ADH (SIADH) which can be diagnosed if the following criteria are fulfilled (Bartter & Schwartz,1967): (1) Hyponatraemia with continued renal excretion of sodium (2) Urine osmolality greater than the appropriate for the tonicity of the plasma, except during an acute isotonic expansion of the extracellular fluid volume (3) Fluid Restriction leads to a rise in plasma sodium concentration and diminished urinary sodium excretion (4) Absence of clinical evidence of fluid volume depletion (5) Normal Renal and adrenal function.

But also IVIG infusions tend to cause pseudohyponatremia through delivery of large amounts of proteins [3], although more recent studies have proved that the alterations in serum sodium levels cannot be explained thoroughly by this mechanism. Indeed, one study reported that hyponatremia, measured by direct ion-selective electrodes, was also present after IVIG infusion.

This "true hyponatremia" was related to osmotic translocation of water from the intracellular to the extracellular (intravascular) space mediated by an increase in intravascular osmolality secondary to sucrose-based IVIG infusion. Possibly IL-6 may also play a crucial role in the pathogenesis of GBS-related hyponatremia [3]. In addition, until recently clinicians have underestimated salt wasting without an underlying CNS disease, because it has been considered to be renal salt-wasting (RSW) due to drug adverse effect (eg, cisplatin), in older people with variable common (eg, hip fracture, pulmonary infections) and other sporadic conditions [3].

Our patient case is unique in that severe hyponatremia preceded neurologic symptoms and the diagnosis of GBS which was developed during its hospital course, contrary to most cases reported in the literature, in which hyponatremia was noted after the establishment of the diagnosis of GBS. It is possible that early changes in the autonomic nervous system triggered by GBS might alter water and sodium balance, preceding symptomatic changes in the peripheral nervous system. In conclusion, close monitoring of further hyponatremia induced by IVIG preparations is suggested when a patient is on GBS therapy.

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