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Case Report

CASE REPORT

Guillain–Barré syndrome hyponatremia: is it SIADH or pseudohyponatremia?

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Abstract

Approximately 5% of hospitalized patients with Guillain–Barré syndrome (GBS) experience SIADH; but pseudohyponatremia has also been reported in patients treated with IVIG. We present a case of a 51-year-old male with GBS who developed acute hyponatremia the day after initiation of IVIG; his sodium levels began to improve within 24 h of completion of IVIG. Differentiating between pseudohyponatremia caused by the IVIG treatment and SIADH caused by GBS was the key to successfully treating this patient. This case exemplifies the importance of pursuing further studies to determine the exact cause of hyponatremia in GBS in order to prevent further neurologic damage to the patient.

INTRODUCTION

While Guillain–Barré syndrome (GBS) with SIADH is well reported in the literature, our case report further examines the less described pseudohyponatremia seen with IVIG use. This case provides educational value as it illustrates the process of differentiating between these two causes of hyponatremia which have significantly different treatment courses. In doing so, further neurologic damage to the patient is prevented and the hyponatremia treated effectively.

CASE PRESENTATION

A 51-year-old male with a history of hypertension and hypothyroidism presented with severe back pain, progressive paresthesias and sudden gait instability 1 week following an upper respiratory infection. His physical examination revealed mild weakness, patchy vibratory and light touch sensory loss, and areflexia of his lower extremities. Lumbar puncture was performed which showed an elevated CSF protein count of 176 mg/dL with a normal white blood cell count. Nerve conduction studies showed a non-length dependent demyelinating polyneuropathy, more than axonal polyneuropathy. These results were consistent with GBS. The patient was initiated on IVIG therapy; his course was complicated by hyponatremia, worsening respiratory status, and dysautonomia resulting in hypertension, urinary retention and small bowel ileus.

Four days after the onset of back pain and paresthesias the patient developed an acute hyponatremia (Na 128 mmol/L) within 24 h of starting IVIG, which was initially felt to be a pseudohyponatremia related to IVIG. Hyponatremia may be associated with moderate to severe hypothyroidism, and so serum TSH levels were confirmed to be within normal limits on the patient's home levothyroxine dose. Clinical suspicion for SIADH grew as the sodium level continued to decrease (in the setting of high urine and low serum osmolality). A free water restriction of one liter daily was enforced. His sodium stabilized

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and reached its nadir at 117 mmol/L. However, he developed orthostatic symptoms and pre-renal acute kidney injury (AKI), therefore, his free water restriction was reversed and he was fluid resuscitated. His sodium and urine osmoles continued to normalize at this time. Within 72 h of improvement in his sodium level, there was also stabilization in the decline of his motor, sensory and autonomic symptoms. At the time of discharge, his hyponatremia and hypertension had resolved, but due to persistent weakness and sensory deficits he was discharged to an outpatient rehabilitation center for physical and occupational therapy.

DISCUSSION

Hyponatremia correlates with the clinical course of GBS and its severity is an indicator of poor prognosis [1]. Approximately 5% of hospitalized patients with GBS experience SIADH; but pseudohyponatremia has also been reported in patients treated with IVIG [2]. Our patient developed hyponatremia the day after IVIG treatment began and it started resolving the day after IVIG was completed. Appropriately treating this patient required differentiating between pseudohyponatremia from IVIG treatment and SIADH caused by GBS.

The relationship between SIADH and GBS has been well described with the median time to onset being 8.8 days [3, 4]. Outside of SIADH, there are two additional sodium derangements to be aware of in the setting of IVIG treatment. First, a true hyponatremia can be seen due to movement of intracellular water to the extracellular compartment caused by the sucrose in the IVIG solution [5]. Secondly, serum sodium can be diluted by increased proteins and lipids from the IVIG solutes causing a pseudohyponatremia with an elevated serum osmole level. We diagnosed SIADH in our patient based on the elevated urine osmoles in the setting of decreased serum osmoles.

The causes of hyponatremia in GBS are multifactorial and it can be a challenge to differentiate the true etiology. This case report illustrates that it would be easy to assume pseudohyponatremia from IVIG based on the timing of sodium derangement, when the patient was in fact developing SIADH. Our experience with this case proves the importance of checking serum osmoles, urine osmoles and consistently monitoring serum sodium to determine the etiology when it would be easy to pre-emptively treat for the wrong etiology. Our patient is unique in that the timing of IVIG and hyponatremia were closely correlated and we were able to distinguish between true hyponatremia from IVIG and SIADH, thus preventing further neurologic damage to the patient.

CONFLICT OF INTEREST STATEMENT

None of the authors has any conflict of interest to disclose.

FUNDING

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ETHICAL APPROVAL

No ethical approval was required.

CONSENT

The patient has provided permission to publish these features of his/her case, and the identity of the patient has been protected.

GUARANTOR

Anna M. Zemke.

REFERENCES

1. Saifudheen K, Jose J, Gafoor VA, Musthafa M. Guillain-Barré syndrome and SIADH. *Neurology* 2011;**76**:701–4. DOI:10.1212/WNL.0b013e31820d8b40.
2. Anandan C, Khuder SA, Koffman BM. Prevalence of autonomic dysfunction in hospitalized patients with Guillain-Barré syndrome. *Muscle Nerve* 2017;**56**:331–3. doi:10.1002/mus.25551.
3. James J, Jose J. Syndrome of inappropriate secretion of antidiuretic hormone preceding Guillain-Barré syndrome. *J Clin Diagn Res* 2017;**11**:OD16–7. doi:10.7860/JCDR/2017/30445.10662.
4. Kloesel B, Hickson LJ. Severe hyponatremia as the initial sign preceding Guillain-Barré syndrome, an acute inflammatory demyelinating polyneuropathy: a case report. *Case Rep Neurol Med* 2013;**2013**:923602. doi:10.1155/2013/923602.
5. Nguyen M, Rastogi A, Kurtz I. True hyponatremia secondary to intravenous immunoglobulin. *Clin Exp Nephrol* 2006;**10**:124. doi.org/10.1007/s10157-006-0416-9.