Case Report

DOI: 10.7860/JCDR/2017/30445.10662

Syndrome of Inappropriate Secretion of Antidiuretic Hormone Preceding Guillain-Barré Syndrome

Internal Medicine Section

JOE JAMES[1], JAMES JOSE[2]

ABSTRACT

Guillain–Barré Syndrome (GBS) is an inflammatory polyradiculoneuropathy which is known to produce syndrome of inappropriate Secretion of Antidiuretic Hormone (SIADH). However, the hyponatremia is usually seen after the onset of weakness. Here, we report a case of SIADH that presented with hyponatremic seizures which preceded the onset of GBS by ten days.

Keywords: Hyponatremia, Polyneuropathies, Seizures

CASE REPORT

A 47-year-old male presented to the emergency department with two episodes of generalized seizures. He had been unwell for the past two days with excessive tiredness and diffuse headache. He also had a history of febrile illness three weeks back, which subsided with paracetamol and amoxicillin. He did not have any prior history of seizures or any other significant illness. On examination, he was afebrile and haemodynamically stable. He was drowsy and disoriented but there were no focal neurological deficits. Investigations revealed severe hyponatremia with serum sodium of 106 mmol/L. Urine sodium was raised at 55 mmol/L (normal range <20mmol/L) and urine osmolarity was 572 mosm/kg of water. Serum cortisol and thyroid function tests were normal. The findings were suggestive of SIADH. Computed Tomography (CT) of head, chest X-Ray and ultrasound of the abdomen were normal and a definite cause of SIADH could not be found out.

Ten-days later he complained of weakness of lower limbs. Over the next three days his upper limbs also became weak. He also developed difficulty in turning in bed. Almost simultaneously, he also complained of numbness in both feet which progressed to involve the fingers. The disability progressed and he became bedridden in five days. There was no bulbar or facial weakness. On examination he was conscious and oriented. There were no cranial nerve palsies. His upper limb had Medical Research Council (MRC) grade 2 power with predominant distal weakness and lower limbs had MRC grade 1 power both proximally and distally. All the limbs were hypotonic and the deep tendon reflexes were absent. Plantar response was bilaterally flexor. There were no sensory deficits on examination.

Blood investigations revealed haemoglobin of 11.9 g/dL, total WBC count of 7,900/mm^3, platelet count 2×10^5/mm^3 and ESR was 55 mm at 1st hour. His plasma glucose was 113 mg/dL. Serum sodium was 124 meq/L and potassium was 5 meq/L. Liver and renal function tests were within normal limits. Urine porphobilinogen was negative. Nerve conduction study showed decreased amplitude of Compound Muscle Action Potential (CMAP) in all tested nerves of the upper limbs. Lower limb motor potentials were not recordable. The Sensory Nerve Action Potentials (SNAP) was not elicitable in the upper and lower limbs. CSF study showed a protein of 95 mg/dL, sugar of 57 mg/dL without any cells. The patient was treated with IV immunoglobulin for five days. Hyponatremia was managed with fluid restriction and tolvaptan 15 mg twice daily and serum electrolytes were serially monitored. Hyponatremia gradually resolved and he was discharged after two weeks. On follow up after one month he showed improvement in the neurological status with grade 4 power of upper limbs and grade 3 power of lower limbs. His serum sodium was 142 meq/L.

DISCUSSION

SIADH is a well recognized complication of GBS. In a prospective study of SIADH in GBS, out of the fifty patients with GBS, 48% had SIADH at some stage of their illness [1]. The median time of onset of hyponatremia was 8.8 days after the diagnosis of GBS. Severe hyponatremia (defined as a serum sodium below 125 mmol/L) was seen in six patients (12%) out of which two had seizures. The authors also noted that severe hyponatremia occurred in severe GBS. The median period needed for correction of hyponatremia was 4.2 days. In none of their cases, SIADH preceded the onset of GBS. In our case, hyponatremia occurred ten days before the onset of GBS. At that time, investigations for the usual causes of hyponatremia were unyielding and the cause of SIADH became obvious only when the patient developed an areflexic LMN weakness consistent with Acute Motor and Sensory Axonal Polyneuropathy (AMSAN). Hyponatremia preceding weakness is common in porphyria [2]. Porphyria was also excluded with repeated negative examination of urine porphobilinogen.

There have been very few cases reports of SIADH preceding GBS [3,4]. The pathogenesis of SIADH in GBS is incompletely understood. One mechanism suggested is damage to the hypothalamic cells causing leakage of ADH into the circulation [5]. Another mechanism may be resetting of the osmoreceptors in the hypothalamus [6]. Of late, interleukin 6 (IL-6) has been implicated in the pathogenesis of SIADH in GBS. IL-6 is an inflammatory cytokine that can augment vasopressin release. IL-6 activates subfornical organ and organum vasculosum of the lamina terminalis stimulating thirst and increases vasopressin secretion from the supraoptic and paraventricular nuclei [7]. It is also established that, the number of IL-6 secreting blood mononuclear cells are increased early during the acute phase of GBS [8].

When SIADH precedes GBS, the cause of SIADH becomes obvious only when the patient develops weakness. In severe hyponatremia with altered sensorium, this weakness may be overlooked and the diagnosis may be missed. A cause of SIADH is often not apparent even after a complete history and adequate investigations. Many cases of such idiopathic SIADH occur in elderly. When the cause of SIADH is not evident as in our case, one must be cautious before a diagnosis of idiopathic SIADH is made, especially if there is an antecedent history of febrile illness, because SIADH may be a prerunner of GBS.

The mainstay of treatment of SIADH is fluid restriction with serial monitoring of serum sodium. Immunoglobulin given for the treatment of GBS can produce pseudohyponatremia. This must be kept in mind while monitoring serum sodium, because unnecessary fluid restriction can exacerbate dysautonomia which usually accompanies GBS.

CONCLUSION

Hyponatremia due to SIADH is common in severe GBS, but very rarely can precede the onset of GBS. All cases of unexplained SIADH with a history of antecedent febrile illness should be carefully assessed for the development of GBS.

REFERENCES

[1] Saifudheen K, Jose J, Gafoor VA, Musthafa M. Guillain-barre syndrome and SIADH. Neurology. 2011;76(8):701-04.
[2] Usalan C, Erdem Y, Altun B. Severe hyponatremia due to SIADH provoked by acute intermittent porphyria. Clin Nephrol. 1996;45(6):418.
[3] Hoffmann O, Reuter U, Schielke E, Weber JR. SIADH as the first symptom of guillain-barre syndrome. Neurology. 1999;53(6):1365.
[4] Ramanathan S, McMeniman J, Cabela R, Holmes-Walker DJ, Fung VSC. SIADH and dysautonomia as the initial presentation of guillain-barre syndrome. J Neurol Neurosurg Psychiatry. 2011;83(3):344-45.
[5] Posner JB. Hyponatremia in acute polyneuropathy. Arch Neurol. 1967;17(5): 530.
[6] Penney MD, Murphy D, Walters G. Resetting of osmoreceptor response as cause of hyponatraemia in acute idiopathic polyneuritis. BMJ. 1979;2(6203):1474-76.
[7] Park SJ, Pai KS, Kim JH, Shin J. The role of interleukin 6 in the pathogenesis of hyponatremia associated with guillain-barré syndrome. Nefrologia. 2012; 32(1):114.
[8] Press R, Ozenci V, Kouwenhoven M, Link H. Non-T(H)1 cytokines are augmented systematically early in guillain-barré syndrome. Neurology. 2002;58(3):476-78.

PARTICULARS OF CONTRIBUTORS:
1. Resident, Department of Internal Medicine, Government Medical College, Kozhikode, Kerala, India.
2. Professor and Head, Department of Neurology, Government Medical College, Kozhikode, Kerala, India.

NAME, ADDRESS, E-MAIL ID OF THE CORRESPONDING AUTHOR:
Dr. Joe James,
Njaralakatt House, Pottangadi Road, West Nadakkav, Kozhikode-673011, Kerala, India.
E-mail: drjoejames@gmail.com

FINANCIAL OR OTHER COMPETING INTERESTS: None.

Date of Submission: **Jun 19, 2017**
Date of Peer Review: **Jul 28, 2017**
Date of Acceptance: **Aug 23, 2017**
Date of Publishing: **Sep 01, 2017**