CASE REPORT

Hyponatraemic Encephalopathy as the Initial Presentation of Guillain-Barre Syndrome

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SUMMARY

This report deals with an elderly lady with Guillain-Barre Syndrome (GBS), who presented with features of unusually severe hyponatraemia. The hyponatraemia was probably due to the syndrome of inappropriate secretion of antidiuretic hormone (SIADH). The hyponatraemia resolved with water restriction and infusion of hypertonic saline; GBS was treated with human immunoglobulin (IVIG). This patient's experience stresses the importance of monitoring serum sodium levels as hyponatraemia has been identified to be a marker of poor prognosis in GBS.

KEY WORDS:
Hyponatraemia, Syndrome of inappropriate antidiuretic Hormone secretion (SIADH), Guillain-Barre syndrome

INTRODUCTION

Hyponatraemia has been reported in 26 to 31% of patients with GBS; usually at the time of maximum motor deficits[1,2]. Herein we present a patient with GBS whose initial manifestations themselves were due to severe hyponatraemia.

CASE REPORT

A 63-year old lady was brought to the hospital by her daughters with complaints of headache, vomiting and altered mental status that began three days earlier. Her medical history was significant for an upper respiratory tract infection ten days before the onset of her symptoms.

Initially the patient complained of headache and had occasional vomiting. She was also lethargic. The following day she was confused, not eating well and was lying down all the time. On the morning of admission the patient was very drowsy and was not responding to call.

On arrival at the hospital the patient was very ill. Blood pressure was 130/80 mm Hg and the pulse was 100 beats per minute and regular. Temperature was normal. Heart, lungs and abdomen were normal. The oxygen saturation was 92% at room air. Blood sugar was 6.9 mmol/L. Electrocardiogram was normal.

The findings were mainly in the nervous system. She was very drowsy and responded weakly to painful stimuli. A focused examination showed her fundi to be normal. The pupils were equal and reactive to light. Neck was supple. There was not much of spontaneous movement in her limbs. The deep tendon reflexes were completely absent with normal plantar response.

Lumbar puncture done within an hour of admission yielded a clear and colourless cerebrospinal fluid (CSF) with a protein content of 0.49 g/L (normal: 0.15-0.45), sugar of 4.1 mmol/L (normal: 2.8-4.2). The CSF cell count was negative. The acid fast bacilli (AFB) smears, Indian ink and Grams stains as well as the CSF cultures were later returned negative.

Meanwhile blood laboratory results revealed severe hyponatraemia at 102 mmol/L (normal: 135-145), potassium 2.5 mmol/L (3.5-5.2), chloride 59mmol/L (96-110), urea 4.5mmol/L (3.0-9.0) and creatinine 53umol/L (44-110). The other blood values including complete blood counts, liver function tests, thyroid hormones, calcium and glucose levels were normal. The serum and urine osmolality were 229 mOsmol/Kg (normal: 278-298) and 222 mOsmol/Kg (normal: 100-1400) respectively.

She tested negative for human immunodeficiency virus (HIV) infection. The Venereal Disease Research Laboratory (VDRL) test and the anti-nuclear factor (ANF) were also negative.

A chest radiograph and a magnetic resonance imaging (MRI) of the brain done a few days later were normal.

She was placed on fluid restriction and infused with 3% hypertonic saline. The infusion rate was adjusted according to her clinical response and serial electrolyte levels obtained at regular intervals. She was more alert the following day when her serum sodium level had had been gradually corrected to 110mmol/L. The power in her extremities was graded at 1/5 in the lower limbs and 2/5in the upper limbs (Medical Research Council grading). Her speech was incoherent.

The hypertonic saline infusion was stopped on the third day when the serum sodium was 125mmolL. The fluid restriction was continued over the next three days till her sodium level normalised.

She was started on gamma globulin (IVIG) therapy on the fourth day of admission. It was infused in a dose of 0.4g/ kg daily for five days.

On the sixth day into admission her daughters were alarmed with her mother's tongue 'dancing away in her mouth'. She had developed marked fasciculation of her tongue indicating bilateral hypoglossal nerve involvement.

This article was accepted: 20 November 2008
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Her management was otherwise largely supportive in the form of physiotherapy and nursing care. On the 26th day she was wheeled home with the power in her limbs being 3/5. There was modest improvement in her speech and ability to eat.

She made gradual recovery thereafter, graduating from wheel chair, walking frame, stick and to independent walking over four months. She also regained her reflexes and speech over the same period. She is well three years later.

DISCUSSION

The diagnosis of GBS was based on the patient's clinical presentation and CSF findings. The rather abrupt onset of limb weakness in association with loss of deep tendon reflexes and subsequent bilateral hypoglossal nerve involvement were consistent with GBS. The antecedent viral illness and good response to immunoglobulin therapy too were in keeping with GBS. Increased CSF protein with few or no cells is characteristic of GBS.

Elecromyographic (EMG) studies were not done in this patient for want of facilities. Although electrodiagnostic features might have supported the diagnosis, they are not mandatory as the diagnosis of GBS is primarily based on clinical and CSF findings [3].

This elderly patient tested negative for HIV infection. But HIV seroconversion simulating GBS would have been a serious consideration if the CSF had exhibited pleocytosis instead of the paucity of cells reported in this patient [3].

The initial presentation of altered mental status due to hyponatraemia was interesting. Although the exact pathogenetic mechanism of hyponatraemia in GBS is not known [1], it has been observed in 26 to 31% of these patients and is usually seen during the course of the illness and at the time of maximum motor deficits[1,2]. GBS presenting with SIADH appears to be rare except for an occasional case report in the English literature [1].

The hyponatraemia observed in this patient was probably due to SIADH, a diagnosis of exclusion. The normal blood pressure, cardiac, renal and thyroid function along with depressed serum osmolality and a urine osmolality of more than 100 mosm/l pointed towards SIADH as the basis of hyponatraemia in her [4,5].

The basic problem in SIADH is water retention due to the kidneys' inability to excrete excess water resulting in dilutional hyponatraemia rather than net loss of sodium from the body[4,5]. Thus the mainstay of treatment is water restriction. On the other hand if the hyponatraemia was of abrupt onset and severe enough to precipitate neurocognitive symptoms, then more aggressive therapy is warranted with hypertonic saline infusion [4,5]. The problem is while under correction of the hyponatraemia harbours the risk of brain herniation, overzealous attempt to attain target sodium levels might lead to central pontine myelinolysis [4,5].

Several formulas have been devised to assist clinicians to manage hyponatraemia[4,5]. These guidelines require frequent monitoring of serum and urine sodium levels which may not be possible in smaller facilities. Thus one has to rely on regular clinical assessment of the patient to modify therapy.

Hyponatraemia has been identified as a marker of poor prognosis in GBS [2]. Clinicians should therefore be aware of this less common but potentially fatal presentation of GBS.

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