

A Case of Persistent Hyponatraemia Due to Reset Osmostat

A G Rohana, MMed (Int Med), A W Norasyikin, MMed (Int Med), Z Suehazlyn, MMed (Int Med), Wong Ming, MRCP, S Norlela, MMed (Int Med), M K Norazmi, MMed (Int Med)

Endocrine Unit, Universiti Kebangsaan Malaysia, Kuala Lumpur

Summary

We report a case of a 65 year old Malay lady with long-standing diabetes mellitus, who presented to our institution with a one month history of worsening neck pain and progressive upper and lower limb weakness. She was stable despite severe hyponatraemia which was initially treated as syndrome of inappropriate anti-diuretic hormone (SIADH). This was consistent with her underlying illness which was concluded as cervical tuberculosis (TB) with spinal cord compression. She underwent decompression and bone grafting. Despite continuous treatment her serum sodium levels remained low. There were no other problems with her adrenals or thyroid. A water loading and hypertonic saline perfusion test was performed and supported the diagnosis of reset osmostat. Her serum sodium remained below the normal range and she was discharged well.

Key Words: Hyponatraemia, SIADH, Reset osmostat, Cervical tuberculosis, Water loading test

Case Report

A 65 year old Malay lady presented to our medical institution with a one month history of worsening neck pain. This was associated with progressive weakness of both upper and lower limbs two weeks prior to her admission. There was no obvious loss of sensation and no urinary and bowel incontinence. She had experienced an unquantifiable loss of weight for a few months but denied loss of appetite. There was no cough or shortness of breath, no fever, no symptoms of thyrotoxicosis and no bowel or urinary symptoms. Premorbidly she was independent.

She has had diabetes mellitus and hypertension for 15 years. There was no significant surgical or family history. She had been taking oral metformin and felodipine. There was no allergy. She was a housewife, a suburban-dweller with no history of tuberculosis (TB)

contact nor any high risk behaviour. She did not smoke nor consume alcohol.

On admission she appeared alert and conscious. She was well hydrated with no obvious respiratory distress. Vital signs were stable with blood pressure of 144/70 mmHg and pulse rate of 90/min. Her cardiovascular, respiratory and abdominal examination revealed no abnormalities. There was reduced tone over both upper and lower limbs with obvious weakness with power of between 2-3/5. There were absent reflexes bilaterally and plantar reflexes were equivocal.

Her blood tests revealed severe hyponatraemia of sodium 112mmol/L, potassium of 4.1 mmol/L, urea of 7.6 mmol/L and creatinine of 29 μ mol/L. Her serum albumin was 33 g/l with normal liver profile. Calculated serum osmolality was 269 mOsm/kg. Haemoglobin(Hb) was 9.7g/dL, white cell count was $8.7 \times 10^9/L$ and platelet $379 \times 10^9/L$.

This article was accepted: 23 September 2006

Corresponding Author: Rohana Abdul Ghari, Department of Medicine, Hospital Universiti Kebangsaan Malaysia, Bandar Tun Razak, 56000 Cheras, Kuala Lumpur

Further investigations included an elevated C-reactive protein (CRP) of 2.99mg/dL and Erythrocyte Sedimentation Rate (ESR) of 84 mm/hr. Serum cortisol was 301nmol/L with good sugar control. HbA1c was 6.9%. A cervical spine radiograph was done which showed subluxation of C5 over C6 with fracture of C6 vertebral body. A subsequent CT scan of the spine revealed sclerosed C5 and C6 vertebral bodies which were reduced in height. There was prevertebral soft tissue swelling with hypodense area, possible abscess collection. Her chest radiograph was normal. An MRI revealed findings consistent with pyogenic spondylodiscitis involving C4-C6 with compression of the spinal cord.

A diagnosis of SIADH was made in view of her spinal cord involvement and she was fluid restricted to approximately 1L/day. There was good urine output and euglycaemia was achieved with regular insulin injections. Three days later she had a sodium level of 128mmol/L, serum osmolality of 279mOsm/kg, urine osmolality of 283mOsm/kg and urine sodium of 31 mmol/L.

She subsequently underwent an emergency decompression with bone grafting. Histopathological examination revealed areas of granuloma formations composed of epithelioid macrophages with central necrosis and positive stain for acid-fast bacilli that was consistent with TB. She was started on anti-TB treatment which was complicated by a mild hepatitis that resolved after adjustments of the drugs. During her hospital stay she experienced one episode of upper gastrointestinal bleed which resolved spontaneously without any surgical intervention. She also had nosocomial pneumonia with septicaemia which resolved with a three weeks course of antibiotics.

Throughout her admission she remained asymptomatic despite persistent euvoelaemic hyponatraemia. The corresponding serum and urine osmolality with urine sodium no longer supported the diagnosis of SIADH. Therefore, the diagnosis of reset osmostat was entertained.

After an overnight fast we performed a water loading test where the patient was given 1L of water to drink within ten minutes. A baseline serum sodium and osmolality, urine sodium and osmolality was measured. 1L of urine was collected after two hours. Her serum osmolality was reduced from 289 to 276 mOsm/kg and urine osmolality showed a change from 290 to 114 mOsm/kg. Serum sodium dropped from 135 to 131mmol/L and urine sodium decreased from 58 to 37 mmol/L. We proceeded with a hypertonic saline load with 345 ml of IV 3% saline given over three hours. The serum osmolality increased from 276 to 291mOsm/kg accompanied by an increase in the urine osmolality from 114 to 326 mOsm/kg. Urine sodium observed a rise from 37 to 129 mmol/L. During her hospital stay she had urine output averaging 1.5-2 L/day with euglycaemia and no other electrolyte abnormalities. These tests indicated a normal response of serum and urine osmolality to volume expansion and serum concentration, excluding the diagnosis of SIADH and supported the diagnosis of reset osmostat.

The patient recovered well and remained hyponatraemic with serum sodium remaining within the range of 125-135 mmol/L. She was discharged well two months later with serum sodium of 126 mmol/L and continued her follow ups with the respective teams.

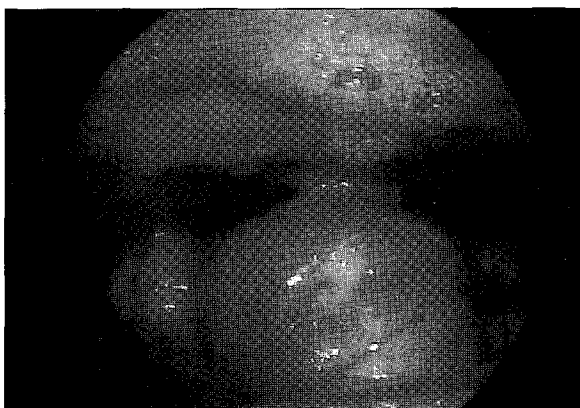


Fig 1:



Fig 2:

Discussion

Hyponatraemia is a common problem throughout both the medical and surgical wards, attributable to many causes and underlying problems. However, we remain vigilant of mortality associated with it, especially concerning sodium levels below 120mmol/L or even higher in the elderly. Therefore, it is intriguing that although our patient presented with severe hyponatraemia (sodium of 112mmol/L) she was stable and maintained good conscious level which suggested a chronic hyponatraemic state. With the initial presentation of spinal cord disease with serum and urine results suggestive of SIADH, the appropriate treatment was given. The patient initially seemed to respond to fluid restriction with serum sodium increasing to 128mmol/L after three days. However, subsequently she remained hyponatraemic despite appropriate treatment with normal levels of serum and urine osmolality. This suggested a resolved episode of SIADH with appropriate treatment unmasking an underlying entity of reset osmostat.

The patient had normal cortisol and thyroid hormone levels. Although her albumin level was low as her hospital stay prolonged, her initial albumin level on admission was normal and could not have influenced her hyponatraemia greatly.

During the water loading test the patient's baseline serum sodium was 135mmol/l, which was at its highest. However, this was taken while the patient's Hb was 8g/dl which was most likely due to chronic disease and prolonged illness. We believe that this may have influenced the actual sodium level.

SIADH is primarily diagnosed by the presence of a hypo-osmolar state with inappropriate urinary response. This differs from reset osmostat, with preserved ability of water excretion, with more than 80% of the loaded water excreted within four hours

during the water loading test¹. Our patient further demonstrated an increase in the serum osmolality with declining urine osmolality which in the presence of inappropriate ADH would have resulted in concentrated urine, i.e., higher urine osmolality and sodium. With the sodium loading test the urine and serum sodium increased. In SIADH urine sodium would have been conserved resulting in a minimal if any rise at all of the urine sodium.

Hyponatraemia occurs in up to a third of long-term care facility and reset osmostat constitutes more than half of that number². The etiology is not clear but together with SIADH, reported cases were described in patients with spinal cord injury, pregnancy, psychosis and other debilitating diseases including tuberculosis, encephalitis, malignancy and malnutrition³, the latter being present in our patient. Neuronal damage to the anterior pituitary causes an abnormal resetting of the hypothalamic osmoreceptors which initiates an inappropriate antidiuretic response perceiving a lower normal serum sodium level³. These cells most likely do not regenerate and, therefore, any damage may be permanent³.

The treatment of reset osmostat and SIADH are different. Hyponatraemia due to SIADH respond quickly to fluid restriction, contrary to reset osmostat in which it worsens. In reset osmostat, patients remain asymptomatic despite serum sodium levels within lower-than-normal range². Therefore it may not be necessary to raise the serum sodium to normal concentrations³.

Hyponatraemia is common and despite our vigilance for its complications and mortality, medical personnel may need to err on the side of caution to avoid overzealous treatment. In asymptomatic patients with severe hyponatraemia, reset osmostat needs to be considered especially if serum sodium levels do not seem to respond to treatment.

References

1. Lipschutz JS, Arieff AI. Reset osmostat in a healthy patient. *Ann Int Med* 1994; 120 (7): 574-76.
2. Chen L, Lim MA, Hwang SJ *et al*. Hyponatremia among the institutionalized elderly in two long-term care facilities in Taipei. *J Chin Med Assoc* 2006; 69(3): 115-19.
3. Kahn T. Reset osmostat and salt and water retention in the course of severe hyponatraemia. *Medicine* 2003; 82 (3): 170-76.