

## Case Report

### **Cerebral salt wasting syndrome in tuberculous meningitis**

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#### **Abstract**

Case of a seventy year old female, who developed cerebral salt wasting syndrome in association with Tuberculous Meningitis is presented.

#### **Introduction**

Cerebral Salt wasting Syndrome (CSWS) is often an unrecognized cause of hyponatraemia and when associated with encephalopathic features it is usually confused with Syndrome of inappropriate anti diuretic hormone secretion (SIADH). Cerebral salt wasting syndrome is characterized by natriuresis, hyponatraemia and volume contraction in response to cerebral pathology.<sup>1</sup> Differentiating it from SIADH is key in managing a patient with CSWS as both are managed paradoxically but present some what similarly.<sup>2</sup>

#### **Case Report**

A 70 year old female presented with complaints of incontinence of urine for four days with fever and drowsiness for last one day. She had a history of significant weight loss for the last six months. On examination she was drowsy and on admission Glasgow coma scale was 7/15. At the time of admission, her haemoglobin was 11.2g/dL (11-14g/dL normal), haematocrit was 32% (35-42% normal), white blood cells were  $11.1 \times 10^9/L$  ( $4-10 \times 10^9$  normal) with neutrophils being 89.2% (40-75% normal). Investigations showed serum sodium was 122 mEq/L, chloride was 94mEq/L, creatinine was 0.4mg/dL, uric acid was 2.3mg/dL and calcium was 6.6mg/dL. Potassium, bicarbonate and blood urea nitrogen were otherwise in their normal ranges.

Urine biochemistry revealed sodium 167mEq/L (30-150 normal). Chest X-ray showed the presence of patchy alveolar infiltrates in both lungs, which were more prominent in the left supra hilar region and left mid lung zone with mild pleural effusion. Ultrasound guided pleurocentesis was done and the aspirate collected was exudative in nature. Pleural fluid culture later grew Mycobacterium Tuberculi. Due to the consistent fever and drowsiness of the patient a Lumbar puncture was performed. The cerebrospinal fluid analysis showed glucose 31mg/dL (50-75mg/dL normal), protein 174mg/dL (15-45mg/dL normal), total leucocyte count 15H (0-06 normal), with lymphocytes 70%.

Magnetic resonance imaging showed leptomenigeal enhancement and infarcts in supra and infra tentorial regions.

A diagnosis of pulmonary tuberculosis along with tuberculous meningitis and SIADH was made. She was put on anti tuberculous therapy with dietary restriction of water and hypertonic saline. Her serum sodium levels did not show any improvement with hypertonic saline and she was continuously losing sodium in her urine. Her serum cortisol levels were 22.6 ug/dl and aldosterone levels were 5.4 ng/dl. Her daily intake and output charting revealed a total of 100-200 ml increase in output (negative balance) however she was hospitalized and fluid was being replaced. The initial impression of hyponatremia was due to decreased intake and probable SIADH due to meningoencephalitis. She was treated with normal saline and the hypertonic saline as well. As her hyponatremia was recurring and her urine Na was still high, it was assumed that natriuresis was due to hypertonic saline and it was discontinued for 48 hours. She still showed no improvement and her condition worsened and it was found that she was actually losing salt. She was then given fludrocortisone (florinef) after which her serum sodium levels improved. Her symptoms due to hyponatraemia subsided and the diagnosis of cerebral salt wasting syndrome was made. She was discharged on anti tuberculous therapy and oral fludrocortisone tablets. She is now in a healthy state of life.

#### **Discussion**

Hyponatraemia (serum levels below 135mEq/L, normal being 136-148mEq/L) has a wide range of causes however in neurologically injured patients it may be due to Syndrome of inappropriate anti diuretic hormone (SIADH) or Cerebral salt wasting syndrome (CSWS).<sup>2,3</sup> Quite frequently in hospitalized cases, hyponatraemia is diagnosed and treated as SIADH or CSWS, yet it remains often undiagnosed.<sup>4</sup>

Cerebral salt wasting syndrome is characterized by increased natriuresis and diuresis in response to disease process in or around the brain.<sup>1</sup> The exact mechanism underlying cerebral salt wasting syndrome remains unclear.<sup>3,4</sup> Its mechanism has been linked with natriuretic peptides, both brain natriuretic peptide and C-type natriuretic peptide have been implicated. It is important for

a clinician to be able to distinguish between SIADH and CSWS as they both present with the same complaint of hyponatraemia and develop under similar circumstances. The main clinical difference between these two conditions is that of the total fluid status of the patient. CSWS leads to a relative or overt hypovolaemia, whereas SIADH is consistent with a normal to hypervolaemic patient.<sup>3,4</sup> As a result, SIADH is treated with fluid restriction which would worsen CSWS as it is treated with fluids and correction of the low sodium. Fludrocortisone (Florinef) a mineralocorticoid improves the hyponatraemia in patients with cerebral salt wasting syndrome.<sup>1,3</sup> Mineralocorticoids enhance sodium reabsorption in the kidney by direct action on distal tubule cells, resulting in expanded extracellular fluid volume.

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We report this patient with tuberculous meningitis in whom the diagnosis of cerebral salt wasting syndrome was made based on volume depletion and high urinary sodium excretion which responded to fludrocortisone therapy and subsided.

### References

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