Case Report

CSW in a Patient with tuberculous meningitis intertiary Care hospital

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ABSTRACT
We present a case of a 61 years old male patient with tuberculous meningitis (TBM) had polyuria, hypernatremia with high fractional excretion of sodium, low serum uric acid with high FEUA & clinically evident dehydration. A diagnosis of cerebral salt wasting (CSW) secondary to TBM was made based on laboratory and clinical parameters. Patient was treated with ATT, fluid replacement with normal saline and fludrocortisone therapy and recovered. We have discussed the case and reviewed the literature review.

Key words: *Cerebral salt wasting, SIADH, tuberculosis, Hyponatremia*

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INTRODUCTION

Hyponatremia secondary to center nervous system (CNS) infection is a well-known entity occurring either due to cerebral salt wasting (CSW) or syndrome of inappropriate antidiuretic hormone (SIADH). CSW results in hyponatremia due to excessive renal loos of sodium in patients with central nervous system insult. Pathologies other than infections afflicting CNS resulting in Hyponatremia includes tumor, stroke or hemorrhage etc. There are several reports of CSW as a cause of hyponatremia in neurological patients[1]. The differentiation between CSW and SIADH is important, because therapy of one may be detrimental to the other[2]. Patients with TBM may have higher frequency of hyponatremia compared to other CNS infections due to leptomeningeal inflammation, hydrocephalous, raised intracranial pressure and ventriculitis[3]. CSW as causing loss of fluid from the body results in weight loss, negative fluid balance and low central venous pressure. Here we are reporting a case of Cerebral Salt Wasting in a patient with TBM responding well to fludrocortisone therapy.

CASE:

A 61 years old male presented in accident & emergency department of a tertiary care hospital with two weeks history of delirium associated with decreased oral intake, generalized body weakness, headache & intermittent feverish feeling. Patient was a known case of hypothyroidism for last twenty years with poor compliance. There were no other significant past medical, surgical & family history. At the time of presentation, the Pulse was 88/min, Blood pressure was 110/70mmHg, afebrile and maintaining saturation at room air. Clinically, the patient had mild dehydration. CNS examination showed a GCS of 12/15 and no signs of meningeal irritation were seen. Knee and ankle jerks were delayed suggestive of significant hypothyroidism due to possible non-compliance. CT brain was normal. Initial laboratory findings at the time of admission showed Hb 9.4g/dL, WBC 13.8, Serum creatinine1.0 mg/dL, Urea 116 mg/dl, Serum Uric Acid 2.3 mg/dl, Serum Potassium 2.8 mmol/L, serum sodium 117 mmol/L. Based on clinical and biochemical parameters, the initial diagnosis of CSW/SIADH secondary to CNS infection was made. CSF analysis couldn't be done as patient's family did not permit.

Patient was empirically treated with antibiotics and antivirals keeping in view the diagnosis of pyogenic meningitis vs viral encephalitis. Further investigations were conducted during hospital stay to confirm the diagnosis that showed high Fractional excretion of sodium (FeNa), High fractional excretion of phosphate (FePO4), high fractional excretion of uric acid (FeUA), high Urine Osmolality and a low serum Osmolality. Brain natriuretic peptide (BNP) levels were not significantly raised. During hospital stay patient poured around 4 to 5 liters of urine per day and was showing clinical signs of volume depletion. A diagnosis of cerebral salt wasting was confirmed based on clinical findings of severe hypovolemia, very high FeNa, low serum uric acid levels with FeUA.

Table1: Patient's lab parameters during hospital stay

Day of admission	S/Sodium	S/Uric acid	I/O
0	117	2.3	1500/3300
3	118		2000/3700
6	121		2900/4000
9	129		3000/3200
12	130	3.1	3500/3700
15	134		3200/3300

MRI brain with contrast was done on the 7th day of admission which showed no communicating hydrocephalus. ATT was started with high suspicion of tuberculous meningitis, antivirals were discontinued, IV normal saline was continued along with oral salt supplementation and fludrocortisone at a dose of 100mcg twice daily was started. Serial serum electrolytes showed gradual improvement in serum sodium levels and urine output gradually decreased to normal. Patient conscious level improved gradually. The fludrocortisone was decreased to 100 mcg per day and was gradually tapered over next 2 weeks and serum sodium level remained normal.

DISCUSSION

Hyponatremia is a common condition seen in neurological diseases. CSW and SIADH both share the common feature of hyponatremia but clinically differentiating both conditions is necessary as treatment of one is opposite to other. Clinically, CSW is a hypovolemic state whereas SIADH is a euvolemic or hypervolemic state. In patients having volume status inconsistent with SIADH but fulfill the criteria of SAIDH, invasive monitoring of CVP and PCWP should be done in ICU settings[4].

Low serum uric acid levels with a high fractional excretion of uric acid is also common to both CSW and SIADH but FeUA gets normalized in SIADH after the fluid restriction and correction of serum sodium whereas it remains elevated in CSWS even though serum sodium starts returning to normal range. Elevated BNP levels may support the diagnosis of CSW[6]. In the above mentioned patient, a diagnosis of CSW secondary to tuberculous meningitis was made based on his severely dehydrated state, Hypernatremia with high FeNa, hypouricemia with High FeUA and correction of serum sodium levels with Normal saline and fludrocortisone therapy. An elevated level of BNP levels would have suggested a diagnosis of CSW but that was not significantly raised in our case.

Table 2: Differential criteria of CSWS and SIADH

Variable	CSWS	SIADH
Weight	↓	↑
Postural hypotension	++	–
Serum osmolality	↓	↓
Serum uric acid	Normal or ↑	↓
Plasma urea	Normal or ↑	↓
Urine sodium	↑↑	↑
Urine volume	↑↑	↓

CONCLUSION

Our Patient was diagnosed with CSW that responded to IV fluids & fludrocortisone hormone therapy for the correction of hyponatremia along with ATT given for the treatment of primary CNS infection.

DISCLOSURE

None declared

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