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**Case Report**

# Cerebral salt wasting in a case of tubercular meningitis in a child

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## ABSTRACT

A 12-year-old female child presented with fever, headache, vomiting since 20 days and convulsions for 1 day. She was unimmunised and BCG scar was absent. Clinical examination showed signs of meningeal irritation, Kernigs sign and Brudzinkins sign with signs of raised intracranial tension. Fundus examination was suggestive of stage 2 papilledema. Her laboratory reports were normal on admission. LP was with hold in view of raised ICT. CT brain was done. S/O meningeal enhancement, mild communicating hydrocephalus with periventricular ooze, extra axial hyper densities in bilateral sylvian fissures along the tentorium (Basal Exudates) On day 3 of admission she had low serum sodium, serum osmolality High urinary sodium. While on lumbar puncture (LP) and cerebrospinal fluid (CSF) examination, CSF protein, and total leukocyte count (predominant lymphocytes) were all increased. On his 5th day of admission, her serum sodium was low and he had a normal urine output. Fluid restriction was tried in order to rule out syndrome of inappropriate antidiuretic hormone secretion (SIADH) but the patient did not respond to it. Keeping in view the above findings, a final diagnosis of tuberculous meningitis leading to cerebral salt wasting syndrome was made. The patient was started on anti-tuberculous therapy (ATT), IV Steroids, anticonvulsants, 3% NaCl and supportive treatment, to which she responded favourably and was later discharged.

**Keywords:** Cerebral salt wasting, Hyponatremia, SIADH, Tubercular Meningitis

## INTRODUCTION

Cerebral salt wasting syndrome is an underreported cause of hyponatremia and is frequently confused with the syndrome of inappropriate antidiuretic hormone (SIADH) secretion. It is characterized by natriuresis, hyponatremia and volume contraction in response to some form of cerebral pathology.[1] Hyponatremia is common electrolyte disturbance in patients after central nervous system disease. In the patients with tubercular meningitis (TBM), there may be higher frequency of hyponatremia compared to other CNS infections due to leptomeningeal inflammation, hydrocephalus, raised intracranial tension and ventriculitis. The syndrome of inappropriate secretion of anti-diuretic harmone (SIADH) is the main attributed cause to this disorder. In some cases, hyponatremia may be due to renal salt wasting and diagnosed as cerebral salt wasting (CSWS). CSWS was first reported by by Peters in 1950, is defined as the occurrence of an elevated diuresis and natriuresis after cerebral injury, leading to hyponatremia and hypovolemia. Despite substantial evidence in animal experiments, its pathophysiology and pertinency remains a question of debate in clinical settings. There is paucity of prospective studies reporting the frequency and predictators of hyponatremia and its prognostic significance in TBM. Severity of meningitis may result in stress response which may result in dysregulation of hypothalamo pituitary adrenal axis. Estimation of natriuretic peptides (atrial, brain derived, c type and dendroaspis) and anti-diuretic hormone has not been helpful because of physiological compensatory response.

Differential diagnosis of this syndrome from SIADH is of paramount importance in managing a patient with cerebral salt wasting syndrome as the management of both conditions is drastically different but their presenting features overlap.[2]

## CASE REPORT

A 12-year-old female child presented to the emergency department with fever, headache and vomiting since 20 days and convulsions for 1 day. She was unimmunised with no BCG scar mark and belonged to low socio economic class. No H/O of rash

No H/O of ear ache or ear discharge, No H/O of weight loss or evening rise of temperature

No H/O of Kochs's or Kochs's contact, No H/O of any weakness or focal neurological deficit, No H/O of any cranial nerve involvement. On examination she had low grade fever, bradycardia (HR-52) hypertension [126/76 mmHg] (99 thtile) and irregular respiration. She had signs of meningeal irritation, neck stiffness, Kernigs and Brudzinkis signs present. Fundus Examination revealed Grade 2 Papilledma. On admission, her laboratory workup showed CBC normal, liver function tests normal, renal function test normal, electrolytes normal, low uric acid of 2.2 mg/dl (reference range: 3.4-7 mg/ dl). LP was withold in view of raised ICT. CT Brain was done. S/O meningeal enhancement, mild communicating hydrocephalus with periventricular ooze, extra axial hyper densities in bilateral sylvian fissures along the tentorium (Basal Exudates). On day 3 she had hyponatremia serum sodium 125 mEq /L. Serum osmolality was 252 mosm/kg (reference range: 285-295 mosm/kg) while his urinary sodium was raised 71 (Normal 15-20 meq/l) urine osmolality was 372 mosm/kg (reference range: 50-1200 mosm/kg). Chest x-ray was normal, Mantoux test was negative. Later, a lumbar puncture (LP) was performed (guarded) and cerebrospinal fluid (CSF) analysis showed a CSF sugar normal 60 mg/dl corresponding serum sugar 105mg/dl, protein raised of 104 mg/dl (normal: <45 mg/dl.

Raised lactate dehydrogenase (LDH) of 63 U/ml (reference range: 2-7 U/ml), a normal total leukocyte count of 2 (reference range: 0-5) with 100% of lymphocytes. CSF culture revealed no growth and the acid fast bacilli (AFB) turned out to be negative. During her admission, she developed a steady fall in serum sodium and on the 3th day of her stay, it was around 125mEq/l inspite of giving 3% hypertonic saline in view of raised intracranial tension. Fluid restriction was tried to rule out SIADH secretion but the patient did not improve on it.

Keeping in view the above laboratory and clinical findings, a final working diagnosis of tuberculosis meningitis and cerebral salt wasting syndrome with complication of raised intracranial tension was made. Child was treated with ATT, IV steroids, anticonvulsant, IV 3% NaCl and supportive treatment. Her serum sodium became normal level within 3 days and also improved her condition drastically and she was later discharged. Thus, a final diagnosis of tuberculous meningitis leading to cerebral salt wasting syndrome was made in this patient and the patient is on regular follow-up and doing well on ATT.

## DISCUSSION

Cerebral salt wasting syndrome or renal salt wasting is mostly seen a few days after a brain injury, in patients with a normal thyroid and adrenal gland function, having a defective kidney sodium transport mechanism that leads to a decreased extracellular volume.[1] Its incidence is underreported, but it is supposed to be one of the major causes of hyponatremia amongst the neurosurgical cases.[2] Although similar in presentation to SIADH, a few factors help in distinguishing between the two; the main one being the effective arterial blood volume, which is decreased in cerebral salt wasting syndrome while increased in SIADH.[3] Since the treatment of both conditions is different, an accurate diagnosis is necessary in order to save precious time, which if not taken into account, can lead to worsening of the condition.[3]

The main diagnostic features of cerebral salt wasting syndrome are a brain lesion and a loss of sodium and chloride by the kidneys without having any stimuli for it.[4] Even though its cause is still not known, researchers have concluded that low sodium in patients with brain disease might be due to cerebral salt wasting syndrome.[5]

Cerebral salt wasting syndrome can also occur without brain disease.[1] Younas et al in their study of 3 patients with cerebral salt wasting syndrome diagnosed their patients based on three parameters; excretion of urinary sodium along with uric acid and a tremendously low serum uric acid.[6] These three parameters were also measured in our case.

Nishio et al reported case of cerebral salt wasting syndrome in middle aged Japanese women who was diagnosed with limbic encephalitis and had increased protein on CSF analysis seen on LP and had presented with psychiatric issues.[7] Celik et al reported two cases of cerebral salt wasting syndrome in children having status epilepticus.[8]

Bettinelli et al studied 110 patients with brain disorders having cerebral salt wasting syndrome and concluded that one of the main underlying cause was found to be TB meningoencephalitis, as seen in our case too.[9] Treatment of cerebral salt wasting syndrome includes fluid along with sodium replacement, which is done via hypertonic saline.[5] Hedge treated his case of cerebral salt wasting syndrome with hypertonic 3% saline which not only improved the patient's consciousness but also his sodium levels, a similar situation to our case.[2]

## CONCLUSION

In conclusion, this case illustrates the point that patients with low serum sodium and some intracranial pathology might be suffering from cerebral salt wasting syndrome. This should also be differentiated from SIADH, as a wrong diagnosis could lead to an inappropriate treatment and might add to the morbidity of patient.

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