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Hyponatremia in Tuberculosis Meningitis: Navigating the Diagnostic Challenges of Cerebral Salt Wasting – A Case Report

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ABSTRACT

A 10 year old female presented with fever, headache and vomiting since four days. Her laboratory workup revealed low serum sodium, serum osmolality. CT scan of the brain was unremarkable, while on CSF examination, protein, and total leukocyte count (predominantly lymphocytes) were increased. CSF cultures were negative, however, GeneXpert detected rifampicin resistant mycobacterium tuberculosis. Due to her high urine output and hyponatremia with sodium of 128 mEq/l, fluid restriction was attempted in order to rule out the diagnosis of SIADH, but the patient was unresponsive to it. Thus, the patient was diagnosed with tuberculous meningitis after further workup, followed by cerebral salt wasting. She was started on anti-tuberculous therapy (ATT), 3% hypertonic saline and flucrocortisone, to which she was responsive, and eventually discharged.

Keywords: SIADH; tuberculous meningitis; cerebral salt wasting; hyponatremia; fludrocortisone.

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INTRODUCTION:

Tuberculosis (TB) represents a worldwide disease epidemic, with an approximate occurrence of 9.6 million new cases in 2014.[1] Tuberculous meningitis (TBM) arises in 1–5% of individuals with TB and is typified by an progressive granulomatous inflammation affecting the basal meninges.[1] Hyponatremia in TBM results from diverse factors such as anorexia, nausea, vomiting, inadequate sodium intake, diarrhea, specific medications, and associated comorbidities.[2] CSW and SIADH both manifest with hyponatremia, but accurate differentiation is vital as their treatments are conflicting.[3] There are numerous case reports on syndrome of inappropriate antidiuretic hormone (SIADH) and cerebral salt wasting (CSW) in TBM.[2-6] CSW can contribute to hyponatremia in patients affected by neurological conditions.[7] CSW and SIADH share comparable features and laboratory indicators, yet distinct treatment approaches, emphasizing the significance of accurate diagnosis and effective management. Diagnosis of CSW involves identifying evidence of hypovolemia, hyponatremia with reduced plasma osmolality, inappropriately increased urine osmolality, elevated urine sodium concentration indicating a negative sodium balance, and low serum uric acid concentration due to urate excretion in urine.[7] Distinguishing between CSW and SIADH is crucial, as treatment for one condition could worsen the other.[3] Here, we present a case of a pediatric patient with TBM that displayed a positive response to fludrocortisone therapy for Cerebral Salt Wasting.

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Case report:

A 10 year old girl presented to us via the emergency department with fever, headache and vomiting since 4 days. On admission, her blood workup revealed a serum sodium of 128 mEq/l, serum potassium of 3.1 mEq/l, while the rest of the electrolytes were within range. CT scan of brain was done, which was unremarkable. Following her lumbar puncture, cerebrospinal fluid (CSF) analysis revealed protein of 141 mg/dL, total leukocyte count of 95/mm3, with 73% lymphocytes, and rbc of 3600/mm, raising the suspicion of tuberculous meningitis. CSF culture revealed no growth while the CSF AFB smear, and CSF gram stain were negative. HbA1C levels, serum cortisol levels and thyroid profile were done and were within normal ranges.

Although she remained vitally stable, she was monitored for any instability, hemodynamic or otherwise. GeneXpert was sent, which was positive for mycobacterium tuberculosis with rifampicin resistance. Upon further inquiry, it was discovered that family history for tuberculosis was positive in her older brother. She was started on multi drug resistant (MDR) anti-tuberculous therapy.

During the course of her admission, her serum sodium levels initially remained low despite treatment with 3% hypertonic saline with a high urine output of 6ml/kg/hr. Her spot urinary sodium was 103 mEq/l, and urine osmolality was 252 mOsm/kg, while her serum osmolality was 266 mOsm/kg. Uric acid levels were 2.6 mg/dl. Fluid restriction was done to rule out the diagnosis of Syndrome of Inappropriate Secretion of ADH, and the patient did not respond to this treatment plan. Based on the above findings, both clinical

and laboratory, the child was diagnosed with tuberculous meningitis and cerebral salt wasting.

The child was managed for electrolyte imbalances with 3% hypertonic saline, as well as treated with mineralocorticoids and started on anti-tuberculous therapy (ATT). Her serum sodium levels increased to a normal level within 7 days, and her clinical condition improved significantly, and she was discharged to regular follow-ups.

The presented case of a 10-year-old girl showed typical symptoms of tuberculous meningitis—fever, headache, and vomiting. However, what sets this case apart is the atypical finding in her blood work; low serum sodium (128 mEq/l) and potassium (3.1 mEq/l); prompting concern for cerebral salt wasting, a rare complication of tuberculous meningitis. Notably, encountering such electrolyte imbalances in pediatric cases with this condition is infrequent, adding complexity to the diagnosis of the etiology of hyponatremia, especially differentiating it from SIADH, and consequently, treatment of this particular case.

DISCUSSION:

In patients with neurological impairment, hyponatremia (serum levels below 135mEq/L, with normal ranges between 136-148mEq/L) may stem from SIADH or CSW, among other potential causes.[5]

Neurological patients can experience hyponatremia due to factors like diuretic use, hypotonic solutions, and comorbidities such as diarrhea. Swift identification and addressing of these factors are crucial. However, our patient did not exhibit these characteristics.[4]

Serum sodium regulates osmolality via ADH and natriuretic peptides. Hyponatremia, below 120mEq/L, or rapid decline causes neurological issues. Tuberculosis meningitis can lead to hyponatremia, often due to cerebral salt wasting (CSW) rather than SIADH. Distinguishing between them is crucial for proper treatment, based on evidence of hypovolemia or euvolemia/hypervolemia.[2]

CSWS requires fluid and sodium replacement, whereas SIADH demands fluid restriction. Both are linked to neurological conditions, sharing features like hyponatremia, urinary Na+ >40 meq/l, and hyperuricemia. SIADH, common in TBM, involves ADH excess and volume retention. CSW, a hypovolemic hyponatremia, triggered by brain natriuretic peptide, increases water and Na+ release, resulting in elevated urinary Na+.[4, 8]

As the case unfolds with the classic presentation of tuberculous meningitis, it emphasizes the importance of thorough investigation even in the absence of typical imaging abnormalities. The identification of rifampicin resistance highlights the evolving challenges in tuberculosis management. Furthermore, the rare occurrence of cerebral salt wasting added complexity, necessitating a nuanced approach to fluid management. Despite the initial resistance to fluid restriction, the combination of anti-tuberculous therapy, 3% hypertonic saline, and mineralocorticoids proved effective, showcasing the intricacies of managing multifaceted complications in pediatric cases.

Distinguishing between SIADH and CSWS hinges on key measures like volume status, osmolarity, urinary sodium and urinary output. SIADH diagnosis entails hyponatremia, low serum osmolality, high urinary osmolality (>100mOsm/Kg), urinary sodium >20mMol/L, and excluding specific causes. For CSW, essential criteria include polyuria, hyponatremia, and excluding secondary causes. Supportive criteria encompass clinical signs of hypovolemia, negative fluid balance, elevated hematocrit/hemoglobin/serum albumin/BUN, and urinary sodium >40 mEq/L or urine osmolality >300 mOsm/L. Elevated osmolarity doesn't favor SIADH diagnosis.[2]

Cerebral salt wasting syndrome is characterized by specific diagnostic hallmarks: the presence of a brain lesion and the renal excretion of sodium and chloride without any discernible triggers.

Syed Ahmad et all in their study of a patient with tuberculous meningitis with cerebral salt wasting noted serum and urine osmolarity, along with urinary sodium and serum uric acid.[6] These aspects were also examined in our study.

YM Tunio et all in their study support the theory that a considerable proportion of patients with tuberculous bacterial meningitis experience hyponatremia. Failing to diagnose and appropriately address hyponatremia through correct protocols and methodologies can result in severe outcomes, including significant neurological complications.[9]

Constant monitoring of serum sodium levels is crucial during hyponatremia correction to avoid hypernatremia. Caution is needed to prevent overcorrection, which could lead to central pontine myelinolysis. Fludrocortisone at doses of 0.1 to 1mg/day can treat CSWS by stimulating sodium and water reabsorption, expanding intravascular volume. Derived from the natural glucocorticoid, fludrocortisone maintains salt and water balance, addressing adrenocortical insufficiency while stabilizing blood pressure.[10]

CONCLUSION:

This case prompts consideration of atypical presentations in tuberculous meningitis, reinforcing the need for a comprehensive diagnostic approach. The successful management of cerebral salt wasting underlines the significance of adapting treatment strategies to address unique complications, ultimately contributing to the positive outcome observed in this patient.

In conclusion, this case underscores the diagnostic challenges in pediatric tuberculous meningitis and the unexpected manifestation of cerebral salt wasting. The integration of GeneXpert testing for rifampicin resistance adds a layer of complexity to tuberculosis management. The successful

resolution through a tailored approach involving anti-tuberculous therapy, hypertonic saline, and mineralocorticoids highlights the importance of adaptability in pediatric care. This report contributes valuable insights into the multifaceted nature of tuberculous meningitis presentations, encouraging vigilance for uncommon complications in clinical practice.

Authors Contribution:
Yousra Nasir Siddiqui: Selecting and writing the entire case report

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