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RESEARCH ARTICLE

CEREBRAL SALT WASTING SYNDROME IN A PATIENT OF SCRUB TYPHUS

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ABSTRACT

Cerebral Salt wasting (CSW) syndrome is a condition which closely mimics SIADH. The differentiation is by the presence of the signs of dehydration in CSW Syndrome. It is important to differentiate between the two as the treatment of CSW syndrome is fluid therapy whereas the treatment of SIADH is fluid restriction.CSW syndrome usually occurs in neurosurgical patients. Here, we present a case of CSW syndrome in a patient with scrub typhus who also had subarachnoid hemorrhage. A physician should be aware of CSW syndrome and should be able to differentiate it from SIADH.

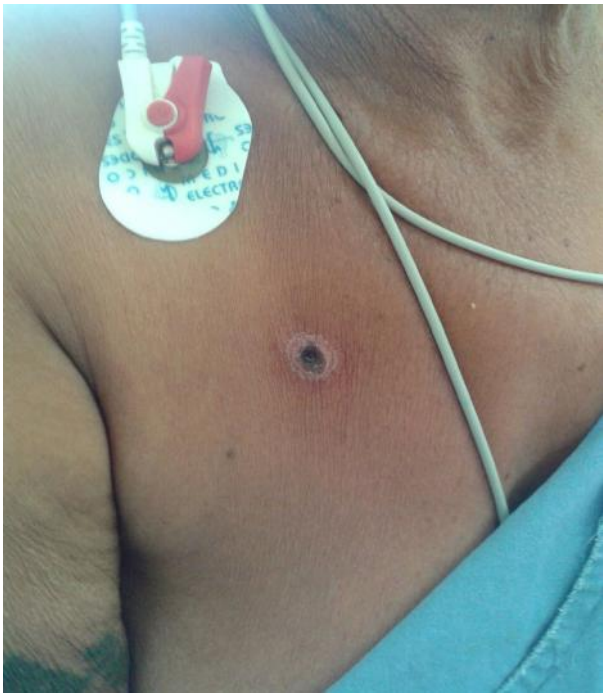
INTRODUCTION

Cerebral Salt wasting syndrome is defined as excessive natriuresis secondary to an intracranial pathology which results in hyponatremia and extracellular fluid loss due to dehydration. CSW syndrome secondary to meningitis, meningoencephalitis and intracranial surgeries are well defined in literature. However CSW secondary to a tropical infection like scrub typhus is not described so far.We report a case of CSW syndrome in a 60 year old lady that occurred following scrub typhus. She had subarachnoid hemorrhage probably developed secondary to thrombocytopenia which resulted in CSW syndrome. Dehydration and elevated BUN made us consider CSW syndrome. Patient recovered with doxycycline, hydration with normal saline and salt supplementation.

Case Report

A 60 year old lady with no premorbid illness presented to our emergency department with history of fever of 1 week duration, altered sensorium and decreased speech output since 2 days. On further enquiry it was revealed that she was admitted at a local hospital and treated for fever, where labs showed thrombocytopenia, which progressively improved within a few days. During her stay in the hospital she developed altered sensorium and decreased speech output and hence was referred to our hospital.

On examination, she had signs of dehydration with a low blood pressure of 86/50 mm of Hg in all four limbs. On CNS examination there were no focal neurologic deficits and examination of other systems were unremarkable. There was presence of an eschar (picture 1) on the right anterior chest wall which made us consider scrub typhus as the etiology, which is a common cause of febrile illness in our geographical region.



Picture 1 Showing the presence of eschar over the right anterior chest wall

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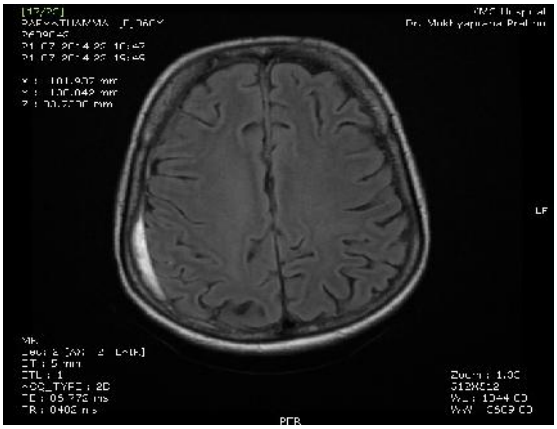
Her hemogram revealed hemoglobin of 12.5gm%(which was later recorded as 10.3 gm% after correcting dehydration the next day), a total leucocyte count of 19000/cu mm of blood with differential count showing Bands and metamyelocytes. In view of high suspicion of scrub typhus, serum IgM for scrub was sent and pending reports she was started on oral doxycycline.

Her biochemical parameters showed serum sodium of 106 meq/l on the day of admission with serum osmolality of 233mOsm, urine osmolality of 413mOsm and urine sodium of 108 meq/l. Thyroid functions and serum cortisol levels were within normal limits. Her Serum uric acid was 1.1mg/dl. Renal functions were normal and liver function tests showed mild elevation of transaminases with raised alkaline phosphatase level. Urine examination and microscopy were normal. Her ECG and ECHO were normal and blood culture sensitivity was sterile. Her serology was positive for Ig M scrub typhus and hence oral doxycycline was continued.

MRI of brain was done in view of altered sensorium and decreased speech output which revealed subarachnoid hemorrhage (picture 2).



Picture 2 Showing Subacute subarachnoid hemorrhage in right temporoparietalconvexity

Neurology and neurosurgery opinion were takenin view of subarachnoid hemorrhage. She was started on oral nimodipine and was managed conservatively with antiepileptic to prevent any seizures.

The presence of dehydration with hyponatremia, high urine sodium excretion and persistence of high urine sodium excretion in spite of correction of hyponatremia made us consider CSW syndrome. She was started on IV hydration with normal saline to correct dehydration and oral salt supplementation.

During her stay in our hospital her sensorium and speech output improved, liver enzymes gradually reverted back to normal and her serum sodium levels gradually improved over 4 days. At discharge her serum sodium levels were normal

DISCUSSION

Cerebral Salt Wasting Syndrome is a much debated topic (1). It was first described in 1957. The clinical and lab features of Cerebral salt wasting Syndrome and SIADH virtually overlap (1, 2). Even though the exact mechanism of CSW syndrome is not described, it proposed to be an increase in the levels of natriuretic peptides. CSW syndrome is considered in a patient with hyponatremia with signs of decreased blood volume (3). It is difficult to distinguish between the two. Blood volume contraction requires isotopic measurement which is not easily available. Moreover, inappropriate urinary sodium excretion is also not clearly defined (2).Distinction is based on decreased volume in CSW and continued excretion of concentrated urine in CSW syndrome in spite of infusion of large volumes of isotonic saline (4).It is important to distinguish between the two as the treatment of both are different, fluid and salt supplementation in CSW whereas, fluid restriction and salt supplementation in SIADH. Some clinicians also recommend mineralocorticoid therapy for CSW Syndrome

Our Patient had hypouricemia and high urine sodium excretion and the urine sodium excretion persisted in spite of correction of hyponatremia, both favoring CSW syndrome (4). Also she had hemoconcentration at the time of presentation which is supported by a rapid fall in hematocrit with correction of dehydration in the absence of any obvious blood loss. Her initial urine sodium was 108mmol/l which remained high even on the third day at 90mmol/l in spite of the correction of sodium from 106 to 122.

It is quite common in Ruptured intracranial aneurysms, craniopharyngiomas and other neurosurgical conditions (5, 6, 7, 8).CSW secondary to a rickettsial disease like scrub typhus is so far not described in literature. Even though this patient had subdural and subarachnoid hemorrhage secondary to thrombocytopenia, the primary condition was scrub typhus which would have been overlooked.

CONCLUSION

A clinician should always keep this entity in mind and diagnose and treat it promptly as it is a close mimic of SIADH which requires fluid supplementation rather than fluid restriction.

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