



A Case Report of Cerebral Salt Wasting Syndrome Secondary to Tuberculosis Meningitis

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Case History

A 35-year-old male presented with fever, nausea, and vomiting since 15 days along right side upper and lower limb weakness and confusion since 5 days. Patient was admitted, he was vitally stable but hypotensive (90/60 mm-hg) and his serial laboratory reports showed low serum sodium (lowest up to 121). Serum uric acid was 1.2 mg/dl (extremely low). On MRI brain scan showed abnormal lepto and pachymeningeal enhancement along bilateral cerebral hemisphere with solitary small acute lacunar infarct in left inferior frontal lobe. Based on clinical presentation and MRI findings under suspicion of tuberculosis meningitis patient was further investigated. On lumbar puncture (LP) and cerebrospinal fluid (CSF) examination, CSF protein (146 mg/dl), Adenosine Deaminase (20 U/L) and total leukocyte count (8 cu/mm) (*predominant lymphocytes* i.e 99%) were all increased. CSF glucose was low (26 mg/dl) He had a high urine output (up to 7.2 L/day). On urine analysis: his urine sodium was 95 mmol/L and urine osmolality was 580 mosm/kg. Blood osmolality turned out to be 240 mosm/kg. Chest x-ray and abdomen – pelvis ultrasonogram was normal ruling out any malignant metastasis. The patient was started on

3% hypertonic saline, mineralocorticoids (i.e Dexomethasone 12 mg/day) and anti-tuberculous therapy (ATT), to which he responded favourably and was later discharged. Patient was kept on ongoing monitoring of body weight, fluid balance, and serum sodium concentration.

Discussion

Cerebral salt wasting syndrome or renal salt wasting is mostly seen a few days after a brain injury having a defective kidney sodium transport mechanism that leads to a decreased extracellular volume.^[3] Its incidence is underreported, but it is supposed to be one of the major causes of hyponatremia amongst the neurosurgical cases.^[2] 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^[5]. 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^[6]. Treatment of cerebral salt wasting syndrome includes fluid along with sodium replacement, which is done via hypertonic saline and newer approach is to treat with saline and mineralocorticoids (fludrocortisone preferred –

0.2 mg/day), which not only improved the patient's consciousness but also his sodium levels, a similar situation to our case

References

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