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Editorial

Evaluation of Prognostic Value of Hyponatremia in TB Meningitis and Acute Bacterial Meningoencephalitis

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One of the most severe forms of tuberculosis (TB) is known as TB meningitis. It is observed to affect the developing countries in a disproportionate manner resulting in significant morbidity and mortality. Meningitis occurs when infection spreads into the subarachnoid space, resulting in an exudative inflammatory response that can be complicated by obstruction of cerebrospinal fluid (CSF) flow and vascular compression and occlusion, with occasional involvement of the cranial nerves in the brain stem. As soon as the infectious droplet nuclei loaded with Mycobacterium tuberculosis are inhaled, a hematogenous dissemination follows that target oxygen-rich areas of the body, including the brain and other parts of the central nervous system (CNS).

Acute hyponatremia is a common finding in patients with TBM. Hyponatremia in TBM may result from the syndrome of inappropriate antidiuretic hormone secretion (SIADH). Pathophysiology of hyponatremia: a crucial role is played by the Serum sodium for maintaining serum osmolality, and hyponatremia is observed to be associated with both normal or any changes (increase or decrease) in osmolality. In normal individuals, serum osmolality usually ranges from 280 mOsm/L to 295 mOsm/L. It is calculated by the following formula: Serum Osmolality = (Serum sodium x 2 + blood glucose/1.8 + blood urea/2.8) mEq/L. In neurological conditions, two major causes of hyponatremia have been suggested - SIADH and CSW. In SIADH, an inappropriate release of ADH or arginine vasopressin contributes to hyponatremia resulting in low serum osmolality and water absorption. This further results in expansion of extra-cellular volume and dilutional hypotonic hyponatremia even in the presence of normal renal sodium handling. The other major cause for hyponatremia i.e. CSW associates with primary natriuresis that results in hypovolemia and sodium depletion even in the absence of a known stimulus and leads to excretion of a large amount of sodium.

Mechanism leading to hyponatremia in TBM - Asymptomatic hyponatremia: patient with asymptomatic hyponatremia along with volume contraction, displays an increased ADH level as a compensatory response. For restoration of intravascular volume, normal saline is required to be administered and free water should be avoided. Once the intravascular volume is restored, the stimulus for release of ADH is eliminated followed by excretion of excess water resulting in correction of hyponatremia. Symptomatic hyponatremia: the management protocol of hyponatremia in TBM relies on the available evidences in other conditions. Urgent treatment of hyponatremia may be lifesaving, although sometimes a proper assessment of the patient may not be possible because of shock, sedation or mechanical ventilation in severe meningitis. If there is no symptomatic improvement after raising serum sodium by 5 mEq/L during first hour, other causes should be checked including imaging.

Concluding, hyponatremia is common in TBM and most often is a result of CSW. At times, border zone infarction is observed that may be contributed by volume contraction associated with CSW. In comparison to patients treated with saline and salt treatment only, Fludrocortisone treatment has shown faster normalization of serum sodium, but polyuria persists.

Limitations: lack of appropriate evaluation and management can lead to grave and permanent neurological consequences, thereby increasing morbidity and mortality. Clinicians should be aware of the implication of sodium imbalance among patients of TBM and differentiate between the various therapeutic options in order to avoid morbidity and mortality.

It is suggested to carry out further studies to develop strategies for management of volume contraction in CSW and also for better understanding of the mechanism of CSW [1-5].

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