Hyponatremia in Older Individuals

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In the yearly Goulstonian lecture in 1936, Robert Alexander McCance defined the physiological and chemical changes induced by experimental salt depletion in normal men effected by the passage of a virtually salt free urine following salt deprivation.1 Subsequently, Winkler and Crankshaw described a new syndrome characterized by renal salt wasting and hyponatremia in three patients with advanced tuberculosis and lung cancer in 1938,2 and this was followed by several reports in quick succession describing disturbances of salt deficit associated with several conditions including heart failure, cirrhosis of liver and renal failure.3-8 Following the description of conditions of inappropriate Antidiuretic Hormone (ADH) secretion in euvolumic patients with bronchial carcinoma in 1957 by Schwartz et al,9 advances in the past few decades have improved our understanding of the previously little understood syndrome.

While the neurologic effects of acute and chronically developing hyponatremia have been recognized, the ideal rate of correction of the salt deficit under hospital settings has been enthusiastically debated over the years but the last word has not been heard on the issue yet.10-15 A fine tuned balance of water is maintained in the human body by the renal counter-current mechanism in close coordination with hypothalamic osmoreceptors controlling the secretion of ADH, which in turn keeps serum sodium concentration within a narrow range of 138-142 mmol/L despite wide variations in water intake. Hyponatremia, defined as serum sodium concentration of less than 135 mmol/L occurs when a defect in the urinary diluting capacity is coupled with excess of water intake and is the most common electrolyte disturbance encountered not only among the older individuals but across all age groups. Underlying all hyponatremic states is a limitation in urinary dilution, most commonly due to inappropriate secretion of ADH despite serum hypo-osmolality.16 The tendency to develop hyponatremia among older persons is a result of senile dysregulation of renal and endocrine systems responsible for sodium and water metabolism in the body.17 It has long been known that ageing impacts the release of vasopressin in response to infusion of hypertonic saline as well as tilting at 60 degrees for 20 minutes.18,19 While rein secretion is attenuated in older persons, so is the renal action of aldosterone thus impairing the sodium retaining ability of the renal system.20

Clinical approach to hyponatremia today is determined by the assessment of volume status of the patient. A hypovolemic hyponatremia is likely due to renal losses due to diuretic abuse or extrarenal losses from the gastrointestinal tract, besides trauma or burns. A hypervolemic hyponatremia is usually due to a chronic disturbance of the renal, hepatic or cardiac function. Commonly encountered causes of euvoemic hyponatremia include SIADH, drugs, psychosis and glucocorticoid deficiency.16 It is prudent to remember that the older patient would not present with the classical symptoms and may come with mild unrelated disturbances which may easily be confused with a senile process by ageist physicians or a more sinister dementia and lead to a battery of ‘non-invasive’ imaging.

Management of hyponatremia involves the knowledge of the cause of the disturbance and the rate of its development, consideration of goals, needs, clinical state of the patient as well as the skill and training of the treating physician. An acutely developing hyponatremia carries a risk of permanent neurological sequelae unless corrected usually with hypertonic saline (3% NaCl) infused at the rate of 1-2 mL/kg/h, while a loop diuretic like furosemide enhances free water excretion. Severe neurological symptoms may merit an infusion rate of upto 4-6 mL/kg/h. Even the use of 29.2% NaCl has been found safe by Soupart and Decaux.21 However, symptomatic patients with chronic disturbance may have adapted to the state and should be treated with more caution and should not be enthusiastically corrected, lest they suffer osmotic demyelination. Older women on thiazide diuretics are known to be susceptible to permanent neurological complications following prolonged hyponatremia or its rapid correction. For management of hyponatremia and SIADH in older subjects, simple administration of oral or parenteral sodium may not be sufficient and fludrocortisone acetate has been advocated in management.22,23 Chronic asymptomatic hyponatremia throws more challenging situations where correction of sodium levels is not mandated. Lithium and demeclocycline have been used in the past for treatment.24 Years of research in the area of AVP receptor antagonists especially the type 2 receptor have yielded vaptans which cause free water loss or aquaresis when used in SIADH or heart failure. Conivaptan and tolvaptan have been the latest addition to the drugs used for treatment of hyponatremia but these are yet not available in Indian markets.25-27

In the current issue of the journal, in a retrospective analysis of 1440 older subjects admitted to the general medical ICU in M.S. Ramiah Hospital, Bangalore over 18 months, MY Rao, U Sudhir, T Anil Kumar, S Saravanan, E Mahesh, K Punith et al report a significantly high number (30%) with a mean age of 72 years (range 60 to 99) had hyponatremia with mean sodium levels of 113.89 mEq/L before correction. Following correction, a mean sodium level of 129.54 mEq/L was achieved in their study.

It remains to be seen if the findings of their study would change the management of hyponatremia and provide benefit to patients at bedside. The study does not look into the details of correction of hyponatremia, however, it would help in sensitizing the physicians towards an early diagnosis of hyponatremia as it may present in a subtle fashion among older persons who present with unrelated symptoms.

References


