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Commentary

Everything the practicing psychiatrist should know about symptomatic hyponatremia – and then some

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Defined as a serum sodium (Na+) concentration below 135 mmol/L, hyponatremia is likely the most prevalent and clinically significant electrolyte abnormality in medical practice, most recently associated with an adverse COVID-19 outcome [1,2]. A working knowledge of how this disorder presents in mental health practice is advantageous for psychiatrists. Most often arising as an adverse reaction to psychopharmacological agents including antidepressants and mood stabilizers, hyponatremia usually develops slowly, leading to subtle symptoms such as lethargy and confusion.

A high index of suspicion for this condition is especially important in the care of elderly patients on such agents, among whom such symptoms may be misattributed to underlying psychological causes. An optimal approach to managing patients on psychopharmacological agents includes obtaining a baseline basic metabolic panel and testing serum sodium (Na+) levels for any changes in clinical status such as impaired cognition or gait instability. Once hyponatremia is identified, slow-onset symptoms readily resolve after appropriate treatment such as discontinuing the responsible agent.

In contrast, the rapid onset of hyponatremia presents with alterations of consciousness including seizures. Acute hyponatremic encephalopathy is a life-threatening neurological emergency which may occur in the following clinical settings:

- acute psychosis associated with the polydipsia-hyponatremia syndrome
- obsessive-compulsive disorders
- water-loading in anorexia nervosa
- atypical adverse reaction to psychopharmacological agents [3]
- suicide attempt by self-induced water intoxication [4]

The prognosis in such cases depends on a timely diagnosis to facilitate definitive treatment to reduce acute cerebral edema. A high index of suspicion by the treating psychiatrist for symptomatic hyponatremia in patients with acute mental status changes facilitates prompt diagnosis of this condition based on expedited laboratory testing. The interval from diagnosis to definitive therapy is crucial, as 'time is brain' regarding irreversible brain damage.

While intravenous hypertonic saline has been recognized as the treatment of choice for acute water intoxication since 1938 [5], controversy has until recently surrounded its optimal mode of administration [6]. Bolus delivery of 100 milliliters of 3% sodium chloride through a peripheral vein has now been shown in a controlled clinical trial to be as safe as and more efficacious than continuous infusion [7]. This intervention reliably increases the serum sodium concentration by 4 to 5 mmol/L in the first five minutes, which promotes the flow of water down an osmotic gradient and out of cells in the central nervous system. Additional boluses may be administered at 10-minute

intervals depending on the patient's clinical response. This strategy has been used safely in emergency departments together with a strong recommendation to initiate treatment without delay such as for neuro-imaging studies [8]. This same approach is the standard of care in the field for athletes with life threatening exercise-induced hyponatremia and in wilderness medicine [9-12].

A spontaneous diuresis of excess free water often occurs after the initial treatment with hypertonic saline during which the serum sodium concentration increases beyond the maximum limits recommended during the correction in cases of non-acute onset hyponatremia (no greater than 10 and 16 mmol/L in the first 24 and 48 hours respectively). Use of the arginine vasopressin analogue desmopressin (1-deamino-8-d-arginine vasopressin; DDAVP) to moderate such higher rates of correction is not necessary in cases where the hyponatremia is acute in onset, as osmotic demyelination syndrome or central pontine myelinolysis as the adaptation of the central nervous system to hypoosmolality has not yet had time to take place [13].

Arginine vasopressin receptor blockers such as tolvaptan have no role in the treatment of symptomatic hyponatremia, although the safety and efficacy of this agent has been demonstrated in treating chronic hyponatremia caused by the syndrome of inappropriate antidiuretic hormone secretion [14]. Conversely, there is no indication for treatment with hypertonic saline in cases of asymptomatic hyponatremia, which should not be used with tolvaptan due to the risk of over-rapid correction.

While the complexities of managing symptomatic hyponatremia fall under the aegis of medical specialists, the opportunity and responsibility for a timely diagnosis to promote a favorable outcome lie with the treating psychiatrist.

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