LETTER

Broca's aphasia as a clinical manifestation of exercise-associated hyponatremia in a non-athlete male

Dear Editor,

A 56-year-old man presented to the emergency department due to headache and mild instability. His clinical condition progressed rapidly to total inability of movement, Broca's aphasia, and mild tremor. He reported a 3-day journey on a motorcycle the preceding week and strenuous activity at his garden the days before admission. He also mentioned 5 to 6 liters of water intake shortly before his presentation. His medical history included only hypertension. Laboratory studies showed an initial serum sodium concentration of 122 mEq/L, while a computerized tomography showed an empty sella turcica without any signs of brain ischemia or edema. Aggressive treatment with weight-based (2 ml/kg) hypertonic solution 2.7 % led to resolution of his symptoms.

Exercise-associated hyponatremia is a well-recognized condition that is associated with acute rather than chronic hyponatremia. The primary mechanism is the overconsumption of fluids, leading to hyponatremia in a dilutional manner enhanced by secondary mediators of ADH secretion, such as non-osmotic stimuli like pain, stress, nausea and Interleukin 6 (IL-6)[1]. Especially, IL-6 may have a crucial role. Research at the molecular level shed light on the cause of increased IL-6 when high levels of IL-6 mRNA were detected in muscle cells of post-exercise biopsy samples[2]. This is interpreted as a capability of contracting skeletal muscle to increase the IL-6 gene expression in response to prolonged activity (over 120 minutes) and release IL-6 in plasma. Connecting the pieces of the puzzle, IL-6 is a potential factor for the syndrome of inappropriate antidiuresis (SIAD)[3]. On the other hand, fluid losses during prolonged activity may decrease the effective intravascular volume and contribute to antidiuretic hormone (ADH) secretion even if low plasma osmolality is present. This response is due to the baroreceptors located in the aortic arch and carotid sinus. Their function to sense changes in volume pressure affects ADH secretion rate by the cells in the paraventricular nuclei, increasing it in case of hypovolemia. Another factor that seems also to have an important role is the osmotically inactive sodium. Athletes who develop severe exercise-associated hyponatremia exhibit inaction of osmotically inactive sodium during exercise, with evidence for reactivation during recovery in some cases. It seems likely that these athletes fail to mobilize osmotically inactive sodium during exercise to buffer the sweat and urinary sodium losses or exhibit inappropriate osmotically inactive circulating sodium[1].

To sum up, this case highlights how severe exercise-associated hyponatremia can develop even in non-athletes. Early diagnosis and appropriate, aggressive treatment of acute symptomatic hyponatremia with severe neurologic symptoms is paramount to the management of hyponatremia regardless of the cause. It is also necessary to maintain a high suspicion in such cases even if they present with uncommon findings, such as those of a stroke.

Keywords: Hyponatremia, syndrome of inappropriate antidiuretic hormone secretion, SIADH, syndrome of inappropriate antidiuresis, SIAD, exercise, aphasia

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Conflict of Interest
TDF reports lecture honoraria from Boehringer Ingelheim, Mylan, Astra Zeneca, Lilly, Recordati, Bausch Health, Servier, and Innovis. The other authors state there is no conflict of interest.

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