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References

1. Spano SJ, Reagle Z, Evans T. Symptomatic hyponatremia presenting at high altitude. *Wilderness Environ Med.* 2014;25:69–74.
2. Ayus JC, Moritz ML. Exercise-associated hyponatremia masquerading as acute mountain sickness: are we missing the diagnosis? *Clin J Sport Med.* 2008;18:383–386.
3. Bennett BL, Hew-Butler T, Hoffman MD, Rogers IR, Rosner MH. Wilderness Medical Society practice guidelines for treatment of exercise-associated hyponatremia. *Wilderness Environ Med.* 2013;24:228–240.
4. Hew-Butler T, Ayus JC, Kipps C, et al. Statement of the Second International Exercise-Associated Hyponatremia Consensus Development Conference, New Zealand, 2007. *Clin J Sport Med.* 2008;18:111–121.
5. Bennett BL, Hew-Butler T, Hoffman MD, Rogers IR, Rosner MH. In reply to Clinical practice guidelines for treatment of exercise-associated hyponatremia. *Wilderness Environ Med.* 2013;24:468–471.
6. Bruso JR, Hoffman MD, Rogers IR, Lee L, Towle G, Hew-Butler T. Rhabdomyolysis and hyponatremia: a cluster of five cases at the 161-km 2009 Western States Endurance Run. *Wilderness Environ Med.* 2010;21:303–308.
7. Hoffman MD, Hew-Butler T, Stuemfle KJ. Exercise-associated hyponatremia and hydration status in 161-km ultramarathoners. *Med Sci Sports Exerc.* 2013;45:784–791.
8. Hoffman MD, Stuemfle KJ, Fogard K, Hew-Butler T, Winger J, Weiss RH. Urine dipstick analysis for identification of runners susceptible to acute kidney injury following an ultramarathon. *J Sports Sci.* 2013;31:20–31.

In Reply to Symptomatic Hypotonic Hyponatremia Presenting at High Altitude

To the Editor:

We would like to thank Doctors Hoffman and Weiss for their critique of our case report. Their letter references 2 valuable publications not cited in our work: the Wilderness Medical Society (WMS) Practice Guidelines for Treatment of Exercise-Associated Hyponatremia (EAH)¹ and an excellent manuscript describing the overlap in clinical manifestations of EAH and acute mountain sickness (AMS).² Neither of these resources articulates an actual case of hyponatremia mistaken for high altitude illness in the clinical setting, and this was the motivation for putting forth our case report. Furthermore, their letter takes to issue our treatment of hypoosmolar hyponatremia with isotonic fluids, which we will address below.

Treatment of hyponatremia can be challenging in a hospitalized patient and is even more challenging in the prehospital setting where diagnostic testing is limited. Although practice guidelines may offer explicit recommendations, they are based largely on expert opinion. Treatment decisions in individual cases often must be based on incomplete information and observed initial responses to therapy. When tailoring the appropriate response to individual patients, clinical uncertainty regarding the diagnosis being treated and the potential influence of anecdotal bias in the guideline development group need to be considered. The critique of our paper makes the assumption that we are reporting a case of EAH. We would like to challenge that assumption.

Our initial concern for altitude-related illness based on the clinical history was soon challenged by the finding of hyponatremia and, subsequently, by the magnetic resonance imaging (MRI), which failed to demonstrate cerebral edema. One could argue that had the low serum sodium been known earlier, an appropriate initial response by Parkmedics would have been hypertonic saline (HTS). However, the Parkmedics have neither the protocol nor the equipment to check prehospital sodium levels in Sequoia and Kings Canyon National Park. In this setting, the authors believe that isotonic saline was appropriate given the subacute onset (>48 hours) of symptoms and the concern, based on the available history, for dehydration. Once in the hospital, the early observation of relatively high-volume, low-osmolality urine cautioned against a more aggressive approach to correction of her hyponatremia at that time.

If EAH has a similar pathophysiology as syndrome of inappropriate antidiuretic hormone (SIADH), as proposed by Bennett et al in the WMS Practice Guidelines,¹ then the very low urine sodium is the most convincing objective evidence to support our position that this case was not one of simple EAH. In SIADH, body water is retained by the kidney at the expense of sodium because of the renal effects of antidiuretic hormone (ADH). One of the hallmarks of the diagnosis of SIADH should be urine sodium that is greater than 40 mEq/L.^{3–5} Our patient's urine sodium was nearly undetectable, indicating her kidneys were holding onto sodium, strongly arguing against an SIADH picture. The recent practice guidelines for the management of EAH do not address urine sodium as part of the initial evaluation, even though urine sodium less than 10 mEq/L suggests a good response to isotonic saline.⁵

Although we did not use invasive monitoring to confirm initial volume status, clinical indicators did not suggest our patient was in a fluid overloaded state.

Emesis can be a stimulus for appropriate ADH secretion, as a clinical precursor for a hypovolemic state. Our patient also had suspected volume losses from reported diarrhea. The history of possible liberal water consumption was not available until hospital day 2, and the accuracy of this is difficult to verify. Performing orthostatic blood pressures to confirm volume status in an intubated patient is not clinically practical. Additionally, exercise more commonly leads to hypovolemia associated with hypernatremia as shown by the 6:1 hypernatremia to hyponatremia case incidence in Boston Marathoners reported in an 8-year case series.⁶ We agree with Siegel et al⁶ that point-of-care testing of serum sodium plays a critical role in differentiating between the two. Furthermore, our patient's MRI on arrival did not show any signs of edema. All these clinical features argue against a picture of classic volume-overload EAH.

The detailed rationale concerning the use of isotonic fluids when EAH is suspected, referenced by Doctors Hoffman and Weiss's response, is a letter to the editor describing the results of 4 case series with an average of 4.5 patients each. The danger of normal saline use in a patient with an unknown sodium level has not been established, and despite longstanding widespread empiric treatment with isotonic fluids by prehospital providers, grave peril from this therapy has not surfaced in the literature. Conversely, missing the diagnosis of hypovolemia in favor of an incorrect clinical impression of EAH could lead to detrimental patient outcomes, especially if only a few milliliters of HTS are administered before a prolonged backcountry transport.⁷ In any case, our patient had an unknown time of onset of her hyponatremia, which may have occurred at any time during the preceding 48 hours of her camping trip. Within 2 hours of her initial serum sodium evaluation, her sodium level had already risen by 4 points. HTS would have corrected her sodium level rapidly, and in the setting of unknown onset her rate of rise would have surpassed the accepted 10 to 12 mg/dL in a 24-hour period.⁸ The alternation between isotonic and hypotonic fluids during her hospital course was intended to limit her serum sodium rise to 8 to 10 mg/dL in each 24-hour period.⁸

In short, we appreciate the discussion made possible by Doctors Hoffman and Weiss's letter and expertise. From the information available to the authors in this case, we initially mistook a presumed case of subacute hyposmolar hyponatremia for high altitude illness, and considered, but ultimately rejected, EAH as a possible etiology along the path to a full patient recovery.

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References

1. Bennett BL, Hew-Butler T, Hoffman MD, Rogers IR, Rosner MH. Wilderness Medical Society. Wilderness Medical Society practice guidelines for treatment of exercise-associated hyponatremia. *Wilderness Environ Med.* 2013;24:228–240.
2. Ayus JC, Moritz ML. Exercise-associated hyponatremia masquerading as acute mountain sickness: are we missing the diagnosis? *Clin J Sport Med.* 2008;18:383–386.
3. Chung HM, Kluge R, Schrier RW, Anderson RJ. Clinical assessment of extracellular fluid volume in hyponatremia. *Am J Med.* 1987;83:905–908.
4. Graber M, Corish D. The electrolytes in hyponatremia. *Am J Kidney Dis.* 1991;18:527–545.
5. Hato T, Ng R. Diagnostic value of urine sodium concentration in hyponatremia due to syndrome of inappropriate antidiuretic hormone secretion versus hypovolemia. *Hawaii Med J.* 2010;69:264–267.
6. Siegel AJ, d'Hemecourt P, Adner MM, Shirey T, Brown JL, Lewandowski KB. Exertional dysnatremia in collapsed marathon runners: a critical role for point-of-care testing to guide appropriate therapy. *Am J Clin Pathol.* 2009;132:336–340.
7. Lipman G. Clinical practice guidelines for treatment of exercise-associated hyponatremia. *Wilderness Environ Med.* 2013;24:466–468.
8. Vaidya C, Ho W, Freyda BJ. Management of hyponatremia: providing treatment and avoiding harm. *Cleve Clin J Med.* 2010;77:715–726.

Spine Protection in the Austere Environment

To the Editor:

We appreciate the opportunity to comment on the excellent Wilderness Medical Society practice guidelines for spine immobilization in the austere environment.¹ We commend the authors for their presentation of the evidence, and we generally agree with their well-considered recommendations. However, we have some concerns, mainly about the proposed algorithm, "Recommendations for spine clearance and immobilization in the austere environment" (Figure 2 of the article).