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Daniel Gutiérrez Sánchez^{a,b}, Juan P. Leiva-Santos^c, María José Macías López^d, Antonio I. Cuesta-Vargas^{b,*}

^a Fundación Cudeca, Arroyo de la Miel, Málaga, Spain ^b Departamento de Fisioterapia, Instituto de Investigación Biomédico de Málaga (IBIMA), Universidad de Málaga, Málaga, Spain

^c Hospital de Manacor, Mallorca, Islas Baleares, Spain ^d Servicio de Nefrología, Hospital Regional Universitario de Málaga, Málaga, Spain

* Corresponding author.

E-mail address: acuesta@uma.es (A.I. Cuesta-Vargas).

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https://doi.org/10.1016/j.nefroe.2018.07.004

Unmeasurable severe hypernatremia: A different way of using the calculated serum osmolality formula

Hipernatremia incalculable severa: una manera diferente de utilizar la fórmula de osmolaridad sérica

Dear Editor:

Hypernatremia is a common electrolyte disorder with increased morbidity and mortality, especially in elderly and critically ill patients. It is most frequently associated with free water losses, but it can also be induced by the administration of hypertonic saline or sodium bicarbonate.^{1,2} The mortality rates for critically ill patients with hypernatremia are as

high as 50%.³ Symptoms from hypernatremia include lethargy, weakness, irritability, seizures, and are as severe as coma.^{4,5}

A 59-year-old Caucasian female presented to the hospital with subarachnoid hemorrhage and severe intracranial hypertension (ICH). The patient required intubation for ventilator support, and started on vasopressors for hemodynamic support. She was also started on hypertonic (3%) saline as an adjunctive therapy for her ICH. On admission, her

[BUN] + [Glucose] Serum Osmolality (calculated) = 2 [Na⁺] + 2.8 18 With [Na+] unmeasurable, we isolated that variable and use the measured serum osmolality, assuming a negligible osmolar gap. We derived this equation to calculate the expected serum sodium concentration when it was unmeasurable : Serum osmolality (measured) _ [BUN] [Glucose] 18 2.8 Expected serum [Na⁺] = 2



Table 1 – Lab parameters during hospitalization.					
Date	Serum osmolality	BUN	Glucose	Na ⁺ (measured)	Na ⁺ (calculated)
5/10/17	394	6	196	>180	191
5/10/17	403	5	220	>180	194
5/10/17	395	5	126	>180	194
5/10/17	387	6	169	>180	188
5/10/17	387	6	194	>180	187
5/11/17	381	8	197	>180	184
5/10/17 determinations made during 5 h intervals.					

serum sodium was 141 mEq/l. Over the next 4 days, despite hypertonic saline having been discontinued, the patient developed severe hypernatremia that was undetectable by the standard assay (ion specific electrode). Her serum sodium was measured as >180 mEq/l. She also developed polyuria with a urine osmolality of 220 mOsm/l, raising the possibility of diabetes insipidus. D5W was started for this patient in combination with scheduled doses of DDAVP to decrease her serum sodium slowly. We had to use the equation (Fig. 1) for serum osmolality to calculate the serum sodium, given the significantly elevated levels, to ensure proper correction. We recognized that the equation is an indirect marker for serum sodium determination, which incorporates blood urea nitrogen and serum glucose concentrations in its calculation. Her peak serum osmolality was 403 mOsm/kg, and it was later lowered to 381 mOsm/kg in a 24-h interval (Table 1). It was not until 30 h into therapy that the serum sodium level became detectable at 173 mEq/l, with a concomitant serum osmolality of 366 mOsm/kg.

In conclusion, using the equation for serum osmolality is an indirect method to calculate the serum sodium level,⁶ which was especially useful in this patient with severe hypernatremia that was unmeasurable (>180 mEq/l) with our standard assay. The applicability of the above mentioned formula allowed us to decreased the patients undetectable serum sodium close to normal range, preventing over-correction and under-correction.^{7,8} We estimate that the patient's serum sodium could have been as high as 194 mEq/l.

Despite these limitations, we were able to indirectly monitor the patient's serum sodium level and adequately correct to goal.^{9,10} The patient survived her hospitalization and was discharged to a rehabilitation unit with improved serum sodium levels.

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Aravindhan Arumugarajah, Christopher Webster, Luis M. Ortega*

Allegheny General Hospital, Division of Nephrology and Hypertension, 320 East North Avenue, Pittsburgh, PA 15212, United States

* Corresponding author.

E-mail address: lortega@ahn.org (L.M. Ortega).

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https://doi.org/10.1016/j.nefroe.2018.02.012