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Original Investigation

Treatment of Hyponatremic Encephalopathy With a 3% Sodium Chloride Protocol: A Case Series

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Background: 3% sodium chloride solution is the accepted treatment for hyponatremic encephalopathy, but evidence-based guidelines for its use are lacking.

Study Design: A case series.

Setting & Participants: Adult patients presenting to the emergency department of a university hospital with hyponatremic encephalopathy, defined as serum sodium level < 130 mEq/L with neurologic symptoms of increased intracranial pressure without other apparent cause, and treated with a continuous infusion of 500 mL of 3% sodium chloride solution over 6 hours through a peripheral vein.

Predictors: Hyponatremic encephalopathy defined as serum sodium level < 130 mEq/L with neurologic symptoms of increased intracranial pressure without other apparent cause.

Outcomes: Change in serum sodium level within 48 hours, improvement in neurologic symptoms, and clinical evidence of cerebral demyelination, permanent neurologic injury, or death within 6 months' post-treatment follow-up.

Results: There were 71 episodes of hyponatremic encephalopathy in 64 individuals. Comorbid conditions were present in 86% of individuals. Baseline mean serum sodium level was 114.1 ± 0.8 (SEM) mEq/L and increased to 117.9 ± 1.3, 121.2 ± 1.2, 123.9 ± 1.0, and 128.3 ± 0.8 mEq/L at 3, 12, 24, and 48 hours following the initiation of 3% sodium chloride solution treatment, respectively. There was a marked improvement in central nervous system symptoms within hours of therapy in 69 of 71 (97%) episodes. There were 12 deaths, all of which occurred following the resolution of hyponatremic encephalopathy and were related to comorbid conditions, with 75% of deaths related to sepsis. No patient developed neurologic symptoms consistent with cerebral demyelination at any point during the 6-month follow-up period.

Limitations: Lack of a comparison group and follow-up neuroimaging studies. Number of cases is too small to provide definitive assessment of the safety of this protocol.

Conclusions: 3% sodium chloride solution was effective in reversing the symptoms of hyponatremic encephalopathy in the emergency department without producing neurologic injury related to cerebral demyelination on long-term follow-up in this case series.

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INDEX WORDS: Hyponatremia; 3% sodium chloride; hypertonic saline; hyponatremic encephalopathy; sodium; electrolyte abnormality; cerebral demyelination; neurologic injury.

Hyponatremia is the most common electrolyte abnormality in the inpatient and outpatient setting.[1,2] Hyponatremic encephalopathy is the most serious complication of hyponatremia.[3-5] Significant risk factors for developing hyponatremic encephalopathy include female sex, hypoxia, and underlying central nervous system disease.[6-9] The symptoms of hyponatremic encephalopathy are largely related to cerebral edema.[3] Hyponatremic encephalopathy constitutes a medical emergency because it might lead to death or permanent neurologic deterioration due to transtentorial herniation or respiratory arrest if untreated.[3,5,10]

According to the recent European Clinical Practice Guidelines, hypertonic saline solution is recommended for the treatment of hyponatremic encephalopathy regardless of whether it is acute or chronic.[11] They acknowledge that "the body of evidence to base recommendations on this topic was limited."[11(pii24)] The guidelines' recommendation for hypertonic saline solution were based on 9 case series that varied widely in regard to the setting, symptoms, severity, duration, and therapy used to treat hyponatremic encephalopathy.[11] According to the guidelines, most case reports used a total of 500 mL of 3% sodium chloride solution. The guidelines recommend using repeated 150-mL boluses of hypertonic saline solution, but they acknowledge that "there is no

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evidence in published research to support this assertion."[11(pii25)] In this article, we evaluated the efficacy and safety of a uniform treatment protocol of 500 mL of 3% sodium chloride solution infused over 6 hours for the management of hyponatremic encephalopathy in the emergency department.

METHODS

Study Participants

This study was conducted from January 1, 1996, through July 1, 2007, in patients older than 18 years presenting to the emergency department at Hospital Professor Alejandro Posadas in Buenos Aires, Argentina, with symptoms of hyponatremic encephalopathy. Patients with hyponatremic encephalopathy were treated with 3% sodium chloride solution according to a protocol recommended by the Argentinian Society of Critical Care Medicine.[12] The protocol consisted of intravenous infusion of 500 mL of 3% sodium chloride solution administered over 6 hours through a large-bore intravenous cannula in conjunction with fluid restriction.

Patients were eligible for the hypertonic saline solution protocol if they had serum sodium levels < 130 mEq/L with advanced signs of hyponatremic encephalopathy, such as headache, nausea, vomiting, mental status changes, delirium, confusion, stupor, tremor, asterixis, seizures, respiratory arrest, noncardiogenic pulmonary edema, or other evidence of increased intracranial pressure without other apparent cause. Patients were not candidates for this hypertonic saline solution protocol if they had overt hypovolemic hyponatremia, hypervolemic hyponatremia, chronic kidney disease (CKD) stage 5, or pseudohyponatremia or were having a spontaneous free-water diuresis following the administration of saline solution.

Therapy was initiated in the emergency department within the first few hours of presentation in all patients. All patients received a total of 500 mL of 3% sodium chloride solution over a 6-hour period. Fluid restriction was instituted following the hypertonic saline solution infusion, with further therapy directed in consultation with the nephrology service. Baseline blood chemistry values were obtained prior to the administration of 3% sodium chloride solution, and serum sodium samples were drawn according to protocol at 3, 12, 24, and 48 hours, or more often at the physician's discretion. An arterial blood gas Pa_{O_2} was recorded when available. Patient demographics, baseline serum chemistry test results, clinical symptoms of hyponatremia, and their duration were recorded. The comorbid conditions assessed were diabetes mellitus, HIV (human immunodeficiency virus) infection, a history of compulsive or uncontrolled alcohol use (alcoholism), chronic obstructive pulmonary disease, CKD stage 3 or 4, hypertension, left ventricular dysfunction, a history of tobacco use (smoking), cerebrovascular accident, acute myocardial infarction, an active neoplasm (cancer), and hypokalemia (potassium < 3.5 mEq/L). Kidney function was assessed at the time of hospital admission with serum creatinine level. The CKD-EPI (CKD Epidemiology Collaboration) creatinine equation was used to calculate estimated glomerular filtration rate. Patients were monitored monthly for 6 months following the episode of hyponatremic encephalopathy in order to assess adverse outcomes, including mortality and neurologic impairment.

All patients who received the hypertonic saline solution protocol were included in the study. Subsequent episodes of hyponatremic encephalopathy were included if the episode occurred more than 6 months after the previous episode following complete neurologic recovery.

The study was approved by the Institutional Review Board of the Hospital Professor Alejandro Posadas.

Statistical Analysis

All data are expressed as mean ± standard error of the mean, median, or proportions as appropriate. The Kolmogorov-Smirnov test was used to verify the normality of the study variables. Patients with a poor outcome (death or major neurologic sequelae) were compared with those without.

The Kruskal-Wallis or nonparametric Friedman 1-way analysis of variance (ANOVA) was used to compare groups, and repeated-measures ANOVA was used to evaluate changes in serum sodium concentrations within the first 48 hours postadmission. Fisher exact test was used for comparison of count variables.

A univariate logistic regression analysis was carried out on the first episode of hyponatremic encephalopathy to determine whether death or neurologic sequelae was a dependent variable on the following potential covariates: sex, age, hypertension, diabetes, left ventricular dysfunction, CKD, hypokalemia, alcoholism, or cancer. Odds ratios with the appropriate 2-sided 95% confidence intervals were reported. All tests were 2 sided, and $P < 0.05$ was considered statistically significant. The analysis was conducted with SPSS, version 19.0, statistical software (IBM).

RESULTS

Study Participants

There were 71 episodes of hyponatremic encephalopathy in 64 individuals, with 58 individuals having 1 episode each, 5 individuals having 2 each, and 1 individual having 3 episodes of hyponatremic encephalopathy. Twenty-nine individuals presented with seizure activity, 10 were stuporous, 5 were comatose, and 2 had neurogenic pulmonary edema. Patient demographics are reported in Table 1. There was equal distribution of men and women, and most (72%) patients were 65 years or older. The main causes of hyponatremia were syndrome of inappropriate secretion of antidiuretic hormone (SIADH; n = 39 [61%]), thiazide diuretics (hydrochlorothiazide; n = 22 [34%]), and severe symptomatic hypothyroidism (n = 3 [5%]).

Comorbid conditions were present in 56 of 64 (88%) individuals. The most common comorbid conditions were hypertension (n = 45 [70%]), a history of left ventricular dysfunction (n = 23 [36%]), diabetes (n = 14 [22%]), smoking (n = 13 [20%]), and cancer (n = 10 [16%]). Four individuals had a history of alcoholism.

Patient Outcome

All individuals experienced symptoms attributable to hyponatremic encephalopathy, which ranged in duration from 4 hours to 15 days (median, 48 hours) prior to admission. Seventy-five percent presented to medical attention within 72 hours of the first symptom of hyponatremic encephalopathy. Baseline serum sodium level for the 71 episodes of hyponatremic encephalopathy was 114.1 ± 0.8 mEq/L and increased to 117.9 ± 1.3, 121.2 ± 1.2, 123.9 ± 1.0, and 128.3 ± 0.8 mEq/L at 3, 12, 24, and 48 hours following the initiation of 3% sodium chloride

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