

Clinical evaluation of hyponatremia and hypovolemia in critically ill adult neurologic patients: contribution of the use of cumulative balance of sodium

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Abstract

Purpose Knowledge of the cumulative balance of sodium (CBS) is important for the diagnosis of salt disorders and water homeostasis and has the potential to predict hypovolemic status in acute neurological patients. However, an extensive application of the use of CBS is still lacking in the intensive care setting, where salt and water homeostasis represents a priority.

Methods Records of consecutive series of acute neurological patients admitted to a neurointensive care unit over a 6-month period were retrospectively reviewed. CBS was calculated at the admission to the Emergency Department. Discrimination between cerebral salt-wasting syndrome (CSWS) and the syndrome of inappropriate antidiuretic hormone secretion (SIADH) was performed on the basis of the classical criteria. Additionally, we used the findings of a negative CBS exceeding 2 mEq/kg for the diagnosis of

CSWS. Two independent clinicians who were blinded to the CBS results performed diagnosis of the causes of hyponatremia and estimated the daily volemic status of the patients on the basis of clinical parameters. Logistic regression analysis was used to determine the independent prognostic factors of hypovolemia.

Results Thirty-five patients were studied for a total of 418 days. Four patients (11.4 %) fitted the criteria of CSWS and three patients (8.5 %) had SIADH. The unavailability of the CBS led to a wrong diagnosis in three of the eight hyponatremic patients (37.5 %). The risk of developing hypovolemia in patients with negative CBS was 7.1 times higher (CI 3.86–13.06; $p < 0.001$). Multivariate analysis revealed that negative cumulative fluid balance, negative CBS >2 mEq/kg, and CVP ≤ 5 cmH₂O were independent prognostic factors for hypovolemia.

Conclusions CBS is likely to be a useful parameter in the diagnosis of CSWS and a surrogate parameter for estimating hypovolemia in acute neurological patients.

Keywords Cerebral salt-wasting syndrome · Syndrome of inappropriate antidiuretic hormone secretion · Cumulative balance of sodium · Hypovolemia

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Introduction

Critically ill adult neurologic patients have a high incidence of salt and water homeostasis disorders [1]. Hyponatremia is one of the most common of these disorders, and when associated with hypotonicity can cause or increase cerebral edema and intracranial hypertension, thus worsening the neurological outcome [2].

The syndrome of inappropriate antidiuretic hormone secretion (SIADH) and cerebral salt-wasting syndrome

(CSWS) are historically considered the two pivotal causes of hyponatremia in neurologic patients [1, 3–6]. The distinction between these two disorders is important since they require different treatments: fluid restriction in SIADH and sodium and volume replacement in CSWS [1, 3]. Up till now the differentiation between these two disorders has been made by measuring extracellular fluid volume (ECF), which is expanded or normal in SIADH, and decreased in CSWS [1, 5–7]. Many parameters are routinely used to evaluate ECF, however, these variables have limited sensitivity and specificity. Even the variables obtained more invasively may be misleading or not continuously available at the bedside, hence distinction between CSWS and SIADH might be difficult [8–11].

In a review published in 2002, Singh et al. [11, 12] emphasized that the clinical diagnosis of CSWS requires a true deficit of sodium evaluated by mass balances, suggesting that a deficit of sodium exceeding 2 mmol/kg body weight implies a contracted ECF volume. Stressing this concept, a diagnosis of CSWS could theoretically be obtained by a negative cumulative balance of sodium (CBS) exceeding 2 mmol/kg body weight given that no other physiological or pathological reasons for sodium excretion are found [11]. Although intriguing, a widespread application of this method in the intensive care setting is still lacking.

The primary purpose of the present study is to investigate the use of CBS to discriminate SIADH from CSWS in critically ill adult neurologic patients by applying the Singh et al. criteria and using a true deficit of sodium as principal parameter for diagnosing. The secondary purpose of this study is to compare the role of the daily sodium mass balance and cumulative balance of sodium with other surrogate markers of hypovolemia in patients admitted to a neurointensive care unit.

Materials and methods

After approval of the study by our hospital's ethics committee (December 2012), we retrospectively reviewed the records of a consecutive series of patients admitted to the neurointensive care unit of our hospital over a previous 6-month period (March–September). In this period we introduced the practice of calculating the daily balance of sodium in patients with critical neurologic or neurosurgical diseases admitted to our ICU.

Inclusion criteria were: the direct admission from an emergency department, age >14 years, the availability of clinical and laboratory data from the admission to the emergency department until the 14th day in the intensive care unit (ICU), and ICU length of stay (LOS) longer than 48 h. Exclusion criteria were: patients with a history of

adrenal gland disease or glucocorticoid therapy within the last year, acute renal failure defined by the specific criteria introduced by the Acute Kidney Injury Network (rapid time course, reduction in urine output defined as <0.5 ml/kg/h for more than 6 h, absolute increase in serum creatinine of ≥ 0.3 mg/dl or percentage increase in serum creatinine of ≥ 50 %) [13], chronic renal failure in stage IV, V or VI as defined by the international guideline of the Kidney Disease Improving Global Outcomes group (patients with a glomerular filtration rate of <30 ml per min) [14], and acute or chronic heart failure (AMI <3 months, NYHA III–IV) [15].

The evaluation of these patients began at the 1st day of admission to the emergency department, was continued in the ICU and ended at the 14th day in ICU or earlier if the patient was discharged from the ICU or died. Age, sex, simplified acute physiology score II (SAPS II) [16], post-resuscitation GCS score, hospital LOS, and ICU and hospital mortality were recorded.

Sodium balance

Natremia, urinary sodium concentration, plasma and urinary osmolality were recorded from the daily routine laboratory data. Blood and urine samples were sent at room temperature to the clinical laboratory of our hospital and analyzed immediately by the indirect potentiometry method (Advia 2400, Siemens Healthcare Diagnostics, Germany). Sodium excretion was measured in 24-h urine samples. Daily sodium balance was evaluated by analysis of intake and output of sodium, whereas the cumulative sodium balance was calculated from all the daily sodium balances. All the volumes, including the infusion solutions, medications as well as nutrition, were considered for the balances. Loss of sodium in sweat and feces was not included in the calculation of sodium balance. Cumulative sodium balance was identified as “negative” when the loss of sodium was higher than 2 mEq/kg [11, 12]. Hyponatremia was identified as a plasma sodium concentration <135 mEq/l, hypo-osmolality as a plasma osmolality <280 mOsm/kg, increased urinary sodium output as a urine sodium concentration greater than 20 mEq/l, increased urinary osmolality as a urine osmolality greater than 100 mOsm/kg, and hypernatremia was defined as a serum sodium level higher than 145 mEq/l [11, 12].

CSWS and SIADH diagnosis

In order to diagnose and distinguish CSWS from SIADH we followed the algorithm described in Fig. 1. The first step of analysis was the identification of patients with acute central nervous system disease in accordance with the inclusion criteria. The second step was the differentiation

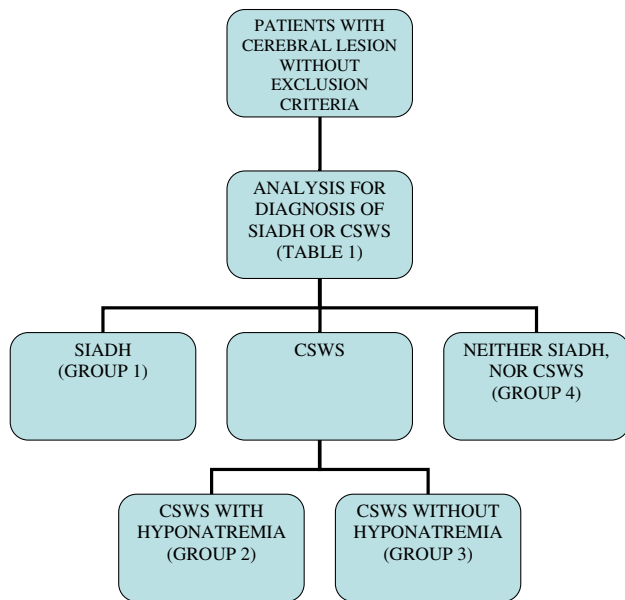


Fig. 1 Algorithm for differential diagnosis of CSWS and SIADH

Table 1 Diagnostic criteria for the diagnosis of SIADH and CSWS [11]

SIADH	CSWS
Hypонатremia (Na <135 mEq/l)	Eunatremia (Na 135–145 mEq/l) or hyponatremia (Na <135 mEq/l)
Plasma hypo-osmolality (plasma osmolality <280 mOsm/kg)	Normal or reduced (<280 mOsm/kg) plasma osmolality
Non-negative cumulative sodium balance (loss of sodium lower than 2 mEq/kg)	Negative cumulative sodium balance (loss of sodium higher than 2 mEq/kg)

of patients according to the diagnosis of SIADH or CSWS by applying the diagnostic criteria as shown in Table 1 [11]. The third and final step was the assessment of natremia in patients with CSWS. Based on this algorithm, we identified four groups of patients: patients with SIADH, patients with CSWS and hyponatremia, patients with CSWS without hyponatremia, and patients without any of these diagnoses. Differences between the groups of patients with and without hypo-osmolar hyponatremia regarding demographics, medical history, ICU and hospital lengths of stay (LOS), mortality, fluid and sodium intake were evaluated.

Clinical evaluation of hypovolemia and CBS

The evaluation of the cumulative balance of sodium as a single surrogate marker of hypovolemia was performed by

comparing it with five clinical signs derived from the retrospective revision of the patients’ charts. According to previous reports and guidelines, the five variables explored were central venous pressure (CVP), mean arterial pressure, hemoglobin and hematocrit values, balance volume and the use of catecholamines [17]. A central venous catheter was placed through the internal jugular vein or subclavian vein and the position of the catheter tip was placed within the right atrium. Hypovolemia was defined as accumulated fluid balance of <60 ml/kg per day or a CVP of 5 mmHg or less, while anemia was defined as a hematocrit level of <33 % [17]. Two independent expert anesthesiologists, blinded to the results of the daily balance of sodium and cumulative balance of sodium, reviewed the five aforementioned clinical signs and assessed the daily volumic status of the patient as euvolemic or hypovolemic. A hypovolemic state was declared only if both the anesthesiologists agreed.

In cases of hyponatremia, the two expert anesthesiologists independently performed a presumptive diagnosis of the causes of hyponatremia.

Statistical analysis

Data are expressed as a mean, with standard deviations for continuous variables and frequencies for categorical variables. In order to make univariate comparisons between normal and hyponatremic groups of patients, Pearson’s chi-squared test was used for categorical variables, a *t*-test for continuous variables and a Wilcoxon–Mann–Whitney for non-parametric variables. Agreement between physicians on the presence or absence of signs of hypovolemia was assessed by the kappa measurement of agreement. Standard formulas were used to calculate sensitivity, specificity, and positive and negative predictive value for each of the clinical parameters.

Clinical signs and parameters evaluated by mass balances rather than excretion rates of sodium (negative cumulative balance, negative fluid balance, use of catecholamine, use of diuretics, CVP ≤5 mmHg, excretion of urinary sodium <25 mEq/l, fractional sodium excretion <1 %, Hb <11.5 g/dl, Htc <33.5 %, MAP >87 mmHg) were collected for primary comparison. In order to make univariate comparisons between hypovolemic subgroups and the presence of one of the variables, logistic regression models were constructed using hypovolemia as the dependent variable and the presence or absence of a possible variable which could explain hypovolemia as the independent variable. Clinical factors found to have a *p* value of <0.20 in the univariate analysis were entered into stepwise multiple logistic regression analysis to determine which were independent prognostic factors for hypovolemia. Effects of selected factors were expressed in

Table 2 Comparison of parameters among subjects with hyponatremia (SIADH and CSWS) and eu-hypernatremic patients

	Population	CSWS without hyponatremia, and neither SIADH nor CSWS (groups 3 and 4)	SIADH and CSWS with hyponatremia (groups 1 and 2)	<i>p</i> value
No. patient (%)	35	28 (80 %)	7 (20 %)	
Gender (M/F)	19/16	15/13	4/3	0.99
Age (years)	51.51 ± 18.86	53.11 ± 18.11	45.14 ± 21.96	0.31
Weight (kg)	74.86 ± 14.26	74.79 ± 14.10	75.4 ± 14.10	0.85
Neurological pathology				
Head injury (HI)	14	11	3	
Stroke	8	8	0	
CNS tumor	2	1	1	0.12
MAV	1	0	1	
HAS aneurysm	9	7	2	
Initial GCS				
Mild (13–15)	10	8	2	
Moderate (9–12)	7	4	3	0.24
Severe (≤8)	18	16	2	
SAPS II	59.46 ± 13.46	60.30 ± 13.18	56.67 ± 15.30	0.44
Daily intake of fluids (l/day)	3.2 ± 1.5	3.17 ± 1.49	3.41 ± 1.64	0.18
Daily intake of sodium (mEq/day)	433 ± 241	420 ± 224	498 ± 228	<0.01
Creatinine clearance (ml/min)	139 ± 64	134 ± 66	154 ± 57	0.01
Mortality in ICU (%)	5 (14.3 %)	5 (14.3 %)	0	0.31
Length of stay in ICU (days)	19.17 ± 11.9	16.82 ± 9.81	28.57 ± 15.50	0.03
Length of stay in hospital (days)	31.62 ± 36.15	23.14 ± 23.73	58.29 ± 55.1	0.08

terms of odds ratios (OR), along with their 95 % confidence intervals (CI). Goodness-of-fit of the model was assessed using the Akaike information criterion (AIC) and McFadden's pseudo R^2 measure [18, 19]. Statistical analyses were computed by R, R Development Core Team [20]. All statistical tests were considered significant for p values of <0.05.

Results

Over a 7-month period, a total of 277 consecutive patients were identified. Of these, 242 patients did not meet the inclusion criteria or were excluded because they did not present with an acute brain lesion, or because they had a stay in ICU shorter than 48 h. Thirty-five patients fulfilled

the inclusion criteria and were followed for a total of 418 days. Patient characteristics at admission are presented in Table 2. Nineteen subjects were male (54.3 %), the median age was 51 years and head injury was the main cause of admission to ICU (40 %). Only thirteen (37 %) patients maintained eunatremia during the ICU observation period. Eight patients (22.8 %) developed hypo-osmolar hyponatremia, while ten patients (28.6 %) developed hypernatremia. In the group of eu-hypernatremic patients, five patients (14.3 %) developed a CSWS without hyponatremia.

Hyponatremic patients: CSWS and SIADH

Eight patients (22.8 %) developed a mild hypo-osmolar hyponatremia. These patients were heterogeneous for

primary diagnosis at admission, demographics and severity of illness at presentation (Tables 2, 3). One of them developed iatrogenic hyponatremia (2.8 %) due to administration of osmotic therapy, while four patients (11.4 %) fitted the criteria of diagnosis of hyponatremic CSWS, and three patients (8.5 %) the diagnosis of SIADH (Table 3). A wrong diagnosis in three of the eight hyponatremic patients (37.5 %) was made by the two expert anesthesiologists because of the unavailability of the CBS.

In all of the eight patients, hyponatremia was rapidly identified, probably due to the frequent check-ups performed during their stay in ICU. The cumulative fluid and balance of sodium values were the main difference between CSWS and SIADH patients. Although fluid restriction or administration was guided by the cumulative balance of salts and fluids (Table 3), the main strategy adopted both for CSWS and SIADH patients was the administration of sodium, which was performed as soon as possible in order to avoid brain damage. The presence of the daily and cumulative balance of sodium, however, simplified the diagnosis and allowed adoption of the best treatment for every patient (Table 3). Figure 2 shows an example of one of the patients with CSWS without hyponatremia, who would have been misdiagnosed by the conventional methods.

There were no significant differences found between the SIADH patients (group 1) and the hyponatremic CSWS patients (group 2) compared to the other two groups [patients with CSWS without hyponatremia (group 3) and the patients without the aforementioned disorders (group 4)], except for the main daily intake of sodium and creatinine clearance, which was higher in groups 1 and 2; and duration in ICU, which was lower in groups 3 and 4 (Table 2). Five patients (14.3 %), all belonging to either group 3 or 4, died in ICU.

Kappa test results

Of the 418 evaluated days, the two physicians agreed only on 235 (56.2 %), which were 78 days (18.7 %) of hypovolemic status and 157 days (37.6 %) of euvolemic and hypervolemic statuses of the patients, respectively. The kappa measurement of agreement was 0.116 (CI 0.025–0.206; $p < 0.05$) for the presence or absence of hypovolemia, indicating a poor agreement between physicians for the diagnosis of volemic status of the patients.

The risk of having hypovolemia was 1.86 times higher in patients with a negative daily balance of sodium (CI 1.01–3.02; $p = 0.02$), and 8.05 times higher in patients with a negative cumulative balance of sodium >2 mEq/kg (CI 3.77–17.18; $p < 0.001$).

Univariate logistic regression analysis

Variables that had p values of <0.05 for the development of hypovolemia were: the use of catecholamines (OR 0.47; CI 0.28–0.79), CVP ≤ 5 mmHg (OR 9.63; CI 5.21–17.78), MAP >87 mmHg (OR 2.28; CI 1.12–4.64), negative cumulative balance of sodium (OR 7.1; CI 3.86–13.05), negative daily balance of sodium (OR 1.82; CI 1.09–3.02), negative cumulative balance of sodium >2 mEq/kg (OR 8.05; CI 3.8–17.2), negative cumulative fluid balance (OR 14.65; CI 7.87–27.30), and negative daily fluid balance (OR 5.82; CI 3.33–10.17) (Table 4).

Multivariate logistic regression analysis

Clinical factors found to have a p value of <0.2 were put in a stepwise multivariate logistic regression for the determination of statistically significant independent prognostic factors for the development of hypovolemia. These factors were: negative cumulative balance of sodium >2 mEq/kg, use of catecholamines, use of diuretics, CVP ≤ 5 mmHg, MAP >87 mmHg, and negative cumulative fluid balance.

Multivariate analysis revealed prognostic factors for the development of hypovolemia negative cumulative fluid balance (OR 6.7; CI 3.4–13.28), negative cumulative balance of sodium >2 mEq/kg (OR 2.74; CI 1.04–7.19), and CVP ≤ 5 mmHg (OR 5.24; CI 2.75–9.99). Evaluation of the full and final model after stepwise inclusion or exclusion of variables showed pseudo R^2 values of 0.30 and 0.29, respectively (Table 5), indicative of a reasonable fit to the data.

Discussion

Our study demonstrates that assessments by bedside observation as to whether hyponatremia is associated with SIADH or CSWS, and evaluation of volemic status of the patients by clinical signs, still remains a dilemma for physicians and intensivists [8, 17]. In the intensive care setting, physicians usually pay particular attention to concentrations of electrolytes and plasma osmolality, checking them frequently. The brain edema consequent to a hypo-osmolar hyponatremia could worsen the outcome of patients with acute neurological disease, therefore normal or hypertonic saline are the most used saline infusions [2, 4]. The frequent usage of this kind of infusion can explain the high mean daily intake of sodium, the high presence of hypernatremic patients (28 %), and the presence of only mild hyponatremia. However, in our study, hyponatremia was found in 22.8 % of the patients admitted to the neurocritical care unit and seems to be related to a longer length

Table 3 Details of patients with hyponatremia

Diagnosis at admission	Age (years)	Weight (kg)	Diagnosis (see Fig. 1)	No. of days of hyponatremia during the 14 ICU days	Plasma sodium (mEq/l)	Plasma osmolality at time of minimum Na (mmol/l)	Study (days)	Daily fluid ^a (ml)	Cumulative fluid ^b (ml)	Cumulative balance of sodium ^a (mEq)	Weighted cumulative balance of sodium ^a (mEq/kg)	Treatment
HSA	70	91	SIADH	2	133	284	13	-1,329	6,364	452	4.97	Fluid restriction of -1152 mL minimal increment of Na of 81 mEq
HSA	62	71	CSWS with hyponatremia	2	133	282	12	-1,110	-7,017	-508	-7.15	Increment of administration of Na of 254 mEq. Increment of basal fluid administration of 2032 mL
TBI	32	63	SIADH	1	134	280	12	164	10,459	1063	16.9	Increment of Na administration of 55 mEq. Fluid restriction of 440 mL
TBI	23	75	CSWS with hyponatremia	1	132	279	14	-150	-2,413	-270	-3.6	Oral sodium supplements
Tumor	67	80	SIADH	1	133	282	14	-230	5,206	294	3.67	Increment of administration of Na of 147 mEq Fluid restriction of 2074 mL
TBI	16	60	CSWS with hyponatremia	1	133	275	7	-170	-2,690	-135	-2.25	Increment of administration of Na of 242 mEq
HSA	46	52	CSWS with hyponatremia	2	133	274	13	-725	-1,684	-201	-3.86	Increment of sodium intake of 106 mEq and fluid of 837 mL

Na CSWS cerebral salt-wasting syndrome, SIADH syndrome of inappropriate antidiuretic hormone secretion, TBI traumatic brain injury, HSA subarachnoid hemorrhage

^a Daily and cumulative fluid and sodium balance performed when the patient was at his sodium nadir

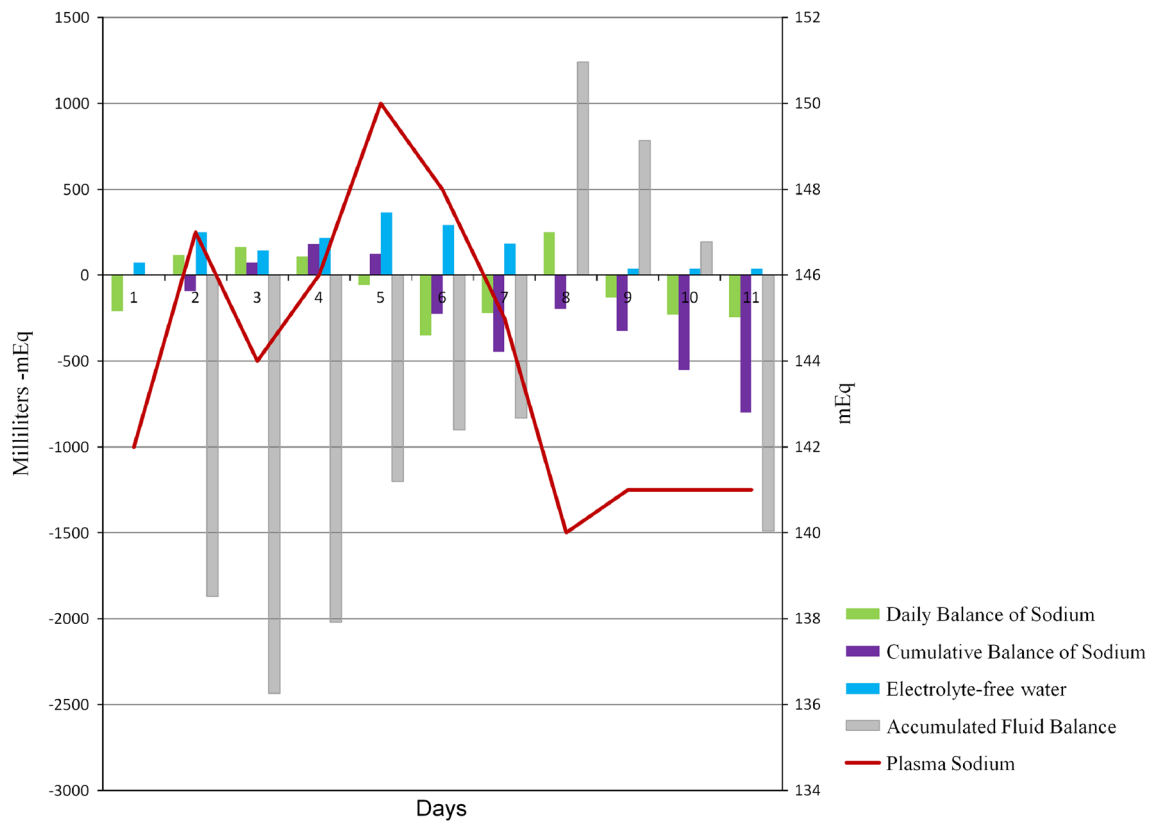


Fig. 2 Example of cerebral salt-wasting syndrome without hyponatremia. Even though there is a loss of sodium as shown by the negative daily balance of sodium and CBS, the movement of fluids

and water seems to compensate CSWS, thus maintaining an adequate plasma sodium concentration

Table 4 Accuracy of the negative cumulative balance of sodium, negative daily balance of sodium and negative cumulative balance of sodium >2 mEq/kg used to predict hypovolemia

Clinical sign	OR (CI)	p value	Sensitivity	Specificity	Positive predictive value	Negative predictive value	LR positive	LR negative
Negative cumulative balance of sodium	7.1 (3.86–13.05)	<0.001	0.37	0.92	0.53	0.86	4.83	0.68
Negative daily balance of sodium	1.82 (1.09–3.02)	<0.02	0.55	0.59	0.24	0.85	1.36	0.76
Negative cumulative balance Na >2 mEq/kg	8.05 (3.8–17.2)	<0.001	0.24	0.96	0.59	0.84	6.33	0.78

OR odds ratio, CI confidence interval, LR likelihood ratio

Table 5 Multivariate logistic regression analysis results

Variable	Coefficient ± SE	Odds ratio	95 % CI	p value	AIC	Pseudo-R ²
Negative cumulative balance of sodium >2 mEq/kg	1.01 ± 0.50	2.74	1.04–7.19	<0.05	277	0.29
Negative cumulative fluid balance	1.90 ± 0.35	6.71	3.39–13.28	<0.01		
CVP ≤5 mmHg	1.66 ± 0.33	5.24	2.75–9.99	<0.01		

The table shows the coefficients of variables included in the model along with their OR and 95 % CI. The AIC and the McFadden pseudo-R² as goodness-of-fit measures of the model are reported

AIC Akaike information criterion, SE standard error, OR odds ratio, CI confidence interval

of stay, as in accordance with previous reports [2, 21]. Even though the hyponatremia was mild, the incidence is impressive because it demonstrates the persistence of the syndromes even in a highly controlled environment such as a dedicated intensive care unit.

Cerebral salt-wasting syndrome with or without hyponatremia represents the most frequent disturbance of salt and water homeostasis in our population, in contrast to other recent findings [22–24]. Although diagnostic criteria for CSWS and SIADH have been clearly defined since the first reports, the greatest part of the studies on distinguishing between these two syndromes have focused on the difference in ECF. Nevertheless, parameters routinely used to evaluate ECF have limited sensitivity and specificity and may be misleading [8–11]. Specialized laboratory tests, such as isotopic red blood cell (RBC) volume measurement or measurement of renin activity in plasma, are not available when the clinical diagnosis of CSW or SIADH has been considered and decisions concerning therapy must be made. Moreover, the majority of the studies show that clinicians use unreliable data such as hyponatremia and the concentration of sodium in urine to conclude if there is salt wasting urine. Electrolytes are not usually measured in patients during the first days of recovery in the ICU, therefore balance data would not be available when the patient manifests hyponatremia [9, 11, 25].

In our study, we were only able to observe a small group of patients. Nonetheless, our study has the merit of exploring the discriminative diagnosis between CSWS and SIADH from a different point of view, performing accurate mass balances of sodium of the patients from admission to the ED until referral to the ICU. The advantage of knowing the sodium status of patients allows clinicians to apply the best treatment in cases of hyponatremia (Fig. 2; Table 3). Moreover, knowledge of the CBS can add information about the volemic status of the patient. To our knowledge, only the study by Carlotti et al. [12] has tried to explore mass balances for sodium accurately during the first 10 days of recovery in 10 children and 12 adults admitted for 10 days to an ICU. However, in none of these patients was the cumulative balance of sodium found to be negative [12]. By studying the sodium balance and changes in plasma volume by using the isotope dilution technique during the 5 days preceding hyponatremia, Wijdicks et al. [26] demonstrated a negative cumulative sodium balance, hyponatremia, and a plasma volume decrease of more than 10 % in 6 of the 21 patients with subarachnoid hemorrhage. In our study, we found nine patients with acute cerebral lesions and negative cumulative mass of sodium exceeding 2 mEq/kg. Four of those patients had a consequent hypo-osmolar hyponatremia suggestive for the diagnosis of CSWS. Our results support the findings by Wijdicks et al. [26] as mentioned above. Furthermore, the

step-wise multivariate logistic regression analysis found that the cumulative balance of sodium >2 mEq/kg, the cumulative balance of fluids, and CVP ≤ 5 mmHg are independently related to hypovolemia.

The major limit of our study is its retrospective nature. Additionally, the procedure of determining the cumulative balance of sodium is time consuming. However, in our opinion, the understanding of mass balance of sodium is essential in the diagnosis of CSWS and SIADH [11]. Besides, it could be a useful parameter which could add information about the volemic status of the patient in the clinical setting of the ICU, where its evaluation may be difficult even for expert anesthesiologists. Our results encourage the idea of refining these findings with further prospective studies, where the main efforts may show mass balance of sodium and water in real time and continuously. It may allow clinicians to evaluate not only the state of salt and water but also to speculate on the volemic status of the patient.

Conclusions

In critical neurology and neurosurgical patients, the use of cumulative balance of sodium is likely to be a simple, economical and efficient tool which may help clinicians to distinguish CSWS from SIADH diagnosis and to improve the comprehension of the volemic status of patients.

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