CLINICAL RESEARCH STUDY

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Characteristics, Symptoms, and Outcome of Severe Dysnatremias Present on Hospital Admission

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ABSTRACT

OBJECTIVE: Dysnatremias are common in critically ill patients and associated with adverse outcomes, but their incidence, nature, and treatment rarely have been studied systematically in the population presenting to the emergency department. We conducted a study in patients presenting to the emergency department of the University of Bern.

METHODS: In this retrospective case series at a university hospital in Switzerland, 77,847 patients admitted to the emergency department between April 1, 2008, and March 31, 2011, were included. Serum sodium was measured in 43,911 of these patients. Severe hyponatremia was defined as less than 121 mmol/L, and severe hypernatremia was defined as less than 149 mmol/L.

RESULTS: Hypernatremia (sodium > 145 mmol/L) was present in 2% of patients, and hyponatremia (sodium < 135 mmol/L) was present in 10% of patients. A total of 74 patients had severe hypernatremia, and 168 patients had severe hyponatremia. Some 38% of patients with severe hypernatremia and 64% of patients with hyponatremia had neurologic symptoms. The occurrence of symptoms was related to the absolute elevation of serum sodium. Somnolence and disorientation were the leading symptoms in hypernatremic patients, and nausea, falls, and weakness were the leading symptoms in hyponatremic patients. The rate of correction did not differ between symptomatic and asymptomatic patients. Patients with symptomatic hypernatremia showed a further increase in serum sodium concentration during the first 24 hours after admission. Corrective measures were not taken in 18% of hypernatremic patients and 4% of hyponatremic patients.

CONCLUSIONS: Dysnatremias are common in the emergency department. Hyponatremia and hypernatremia have different symptoms. Contrary to recommendations, serum sodium is not corrected more rapidly in symptomatic patients.

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KEYWORDS: Emergency department; Hypernatremia; Hyponatremia; Sodium; Symptoms; Treatment

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Dysnatremias are the most common electrolyte disorders in hospitalized patients and are associated with adverse outcomes.[1-9] Despite this, almost no studies focusing on hyponatremia and hypernatremia have been performed in the population presenting to the emergency department. Mohammed et al[10] studied the prevalence of hyponatremia in patients who were admitted to the emergency department with uncontrolled heart failure, and Renneboog et al[11] investigated the incidence of falls among patients with hyponatremia in the emergency department. Only 1 retrospective

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study has examined the prevalence of hyponatremia in the emergency department.[12]

No studies have investigated the prevalence and characteristics of hypernatremia in the emergency department or systematically evaluated the symptoms of dysnatremias in adult patients in the hospital. It is unclear how current recommendations for the correction of hyponatremia and hypernatremia are used in daily practice.

We aimed to evaluate the prevalence of severe hyponatremia and hypernatremia in the emergency department, the prevalence of symptomatic dysnatremias, the nature of symptoms, and the correction rate of dysnatremias in patients who are hospitalized with severe dysnatremias.

CLINICAL SIGNIFICANCE

- Among patients presenting to the emergency department, 10% had hyponatremia and 2% had hypernatremia.
- Neurologic symptoms were common in patients with severe hyponatremia (68%) and hypernatremia (38%).
- A significant portion of patients were untreated or inadequately treated, and 10% to 15% of patients were overcorrected.

MATERIALS AND METHODS

All patients in whom serum sodium was measured at the department of emergency medicine of a university hospital between April 1, 2008, and March 30, 2011, were included in this retrospective study. In patients who were admitted to the emergency department more than once, only the first admission was considered for the analysis.

Hyponatremia was defined as a serum sodium level less than 135 mmol/L, and hypernatremia was defined as a serum sodium level greater than 145 mmol/L. Severe hyponatremia was defined as a serum sodium level less than 121 mmol/L, and severe hypernatremia was defined as a serum sodium level greater than 149 mmol/L.

In all patients with severe hyponatremia or severe hypernatremia, we gathered the following data on admission to the emergency department from the patient administration database of the local emergency department (Qualicare Office, Medical Database Software, Qualidoc AG, Bern, Switzerland): serum—sodium, potassium, osmolality, C-reactive protein, creatinine, bilirubin, lactate, thyroid stimulating hormone, and hemoglobin; urine—sodium and osmolality. All information on the course of serum sodium levels for the 3 days after admission was included in our analysis.

The following was documented for all patients: demographic data, category of admission diagnosis (at the discretion of the physician), transfer to an intensive care unit, date of death, and Glasgow Coma Scale score on admission. We also reviewed patient charts for symptoms of hyponatremia and hypernatremia, defined as follows:[1,8,13,14] weakness, cramps, seizures, somnolence, coma, agitation, restlessness, headache, disorientation, confusion, nausea, emesis, vertigo falls, and syncope. These symptoms were considered related to dysnatremias only if they could not be explained by a different underlying disorder.

Statistical Methods

The results are presented as mean and standard deviation or median and first and third quartiles. Correlations, *t* tests, chi-square tests, and Mann-Whitney *U* tests were computed using STATISTICA 9.1 (StatSoft Inc, Tulsa, Okla). The study was approved by the ethics committee of the Canton of Bern, Switzerland.

RESULTS

Between April 1, 2008, and March 30, 2011, a total of 77,847 patients were admitted to the emergency department. Serum sodium was determined in 43,911 patients. The mean age of patients was 53 years (standard deviation, 20); 18,685 (43%) were women, and 25,226 (57%) were men. Hyponatremia (serum sodium < 135 mmol/L) was found in 4503 patients (10%), and hypernatremia (>145 mmol/L) was found in 678 patients (2%). The lowest serum sodium measured was 97 mmol/L, and the highest serum sodium measured was 177 mmol/L. A total of 74 patients (0.2%) were admitted with severe hypernatremia (>149 mmol/L), and 168 patients (0.4%) were admitted with severe hyponatremia (<121 mmol/L) (**Figure 1**).

Severe Hyponatremia

We identified 168 patients with severe hyponatremia with a serum sodium less than 121 mmol/L. A total of 121 patients (72%) presented to the emergency department for medical reasons, and 47 patients (28%) presented to the emergency department for surgical reasons. The details of admission characteristics are shown in **Table 1**.

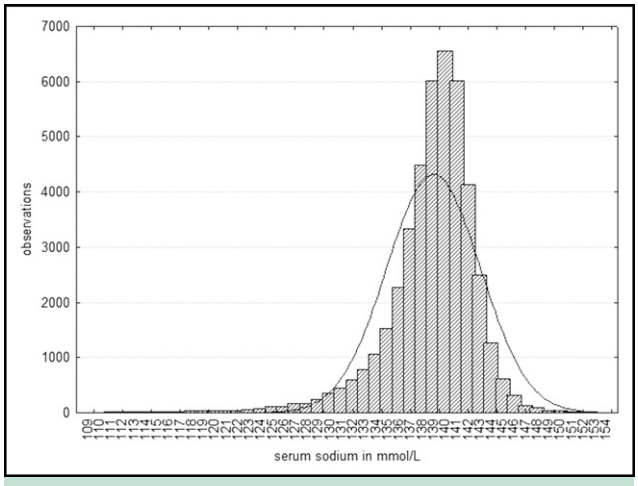

Figure 1 Distribution of serum sodium on admission to the emergency department.

Table 1 Categories of Admission Diagnosis for Patients with Severe Hyponatremia or Hypernatremia: More Than 1 Category Can Apply to 1 Patient

	Hypernatremia (N = 74)	Hyponatremia (N = 168)
Age (y)	58 (SD 23)	64 (SD 16)
Sex (male)	46 (62%)	86 (51%)
Symptomatic dysnatremia	20 (38%)	89 (64%)
Type of admission		
Cardiac	5 (7%)	5 (3%)
Pulmonary	7 (9%)	13 (8%)
Gastrointestinal	4 (5%)	12 (7%)
Hepatologic	1 (1%)	6 (4%)
Gynecologic	0 (0%)	2 (1%)
Renal/urologic	6 (8%)	63 (38%)
Neurologic	11 (15%)	81 (48%)
Endocrine	3 (4%)	4 (2%)
Trauma	10 (14%)	12 (7%)
Psychiatric	3 (4%)	2 (1%)
Intoxication	6 (8%)	0 (0%)
Oncologic	3 (4%)	7 (4%)
HIV	2 (3%)	0 (0%)
Dermatologic	0 (0%)	3 (2%)
Unknown	21 (28%)	27 (16%)

HIV = human immunodeficiency virus; SD = standard deviation.

Serum sodium on admission was 117 mmol/L (Q1: 114, Q3: 119), with a minimum of 97 mmol/L and a maximum of 120 mmol/L. Serum osmolality was 245 mosmol/kg (Q1: 239, Q3: 254) and correlated significantly with admission serum sodium ($R = 0.24$, $P < .05$). Creatinine on admission was 66 μmol/L (Q1: 48, Q3: 96), with a maximum of 795 μmol/L, and urine osmolality was 332 mosmol/kg (Q1: 211, Q3: 507). A total of 24 patients (14%) were taking thiazide diuretics, 12 patients (7%) were taking aldosterone antagonists, 34 patients (20%) were taking opioids, and 43 patients (26%) were taking psychotropic drugs. A detailed overview of medications on admission is shown in **Table 2**.

Symptoms of Severe Hyponatremia. Information on symptoms was available for 140 patients (83%) with severe hyponatremia; 89 patients (64%) had neurologic symptoms. Of these 89 patients, 29 (33%) had 1 symptom, 31 (35%) had 2 symptoms, 18 (20%) had 3 symptoms, 5 (6%) had 4 symptoms, 4 (4%) had 5 symptoms, 1 (1%) had 6 symptoms, and 1 (1%) had 7 symptoms. The most common symptoms on presentation were nausea in 34 (38%) patients, falls in 27 patients (30%), and weakness in 25 patients (28%). **Figure 2** shows the details of the symptoms of severe hyponatremia. The Glasgow Coma Scale score was 15 (Q1: 15, Q3: 15) on admission and did not correlate with serum sodium on admission. Serum sodium was significantly lower in patients who were symptomatic (117 [Q1: 112, Q3: 119] vs 118 [Q1: 116, Q3: 120] mmol/L, P = .03).

Correction of Hyponatremia and Outcome. Data on serum sodium on the day after emergency department admission were available for 144 patients (86%). Specific measures to correct hyponatremia were not given to 7 patients (4% of all patients with severe hyponatremia). Four of these patients had documented symptomatic hyponatremia.

Table 2 Medications on Admission to the Emergency Department of Patients with Severe Hyponatremia and Hypernatremia

Medication	Hypernatremia Overall (N = 74)	Symptomatic Hypernatremia (N = 20)	Hyponatremia Overall (N = 168)	Symptomatic Hyponatremia (N = 89)
ACEI	5 (7%)	3 (15%)	32 (19%)	21 (24%)
AT_1 receptor blocker	4 (5%)	2 (10%)	22 (13%)	10 (11%)
β-blocker	7 (9%)	2 (10%)	47 (28%)	27 (30%)
Calcium channel blocker	0 (0%)	0 (0%)	14 (8%)	9 (10%)
Proton pump inhibitor	8 (11%)	4 (20%)	57 (34%)	34 (38%)
Steroids	3 (4%)	3 (15%)	15 (9%)	10 (11%)
Loop diuretic	8 (11%)	3 (15%)	21 (13%)	10 (11%)
Thiazide	2 (3%)	1 (5%)	24 (14%)	16 (18%)
Spironolactone	4 (5%)	1 (5%)	12 (7%)	8 (9%)
Statin	1 (1%)	1 (5%)	25 (15%)	14 (16%)
NSAID	5 (7%)	3 (15%)	7 (4%)	4 (4%)
Antiaggregant	11 (15%)	4 (20%)	45 (27%)	29 (33%)
Opioid	9 (12%)	6 (30%)	34 (20%)	21 (24%)
Antibiotic	5 (7%)	4 (20%)	10 (6%)	5 (6%)
Thyroid hormone	0 (0%)	0 (0%)	13 (8%)	10 (11%)
Oral antidiabetic	1 (1%)	1 (5%)	12 (7%)	8 (9%)
HIV medication	2 (3%)	1 (5%)	0 (0%)	0 (0%)
Psychotropics	13 (18%)	4 (20%)	43 (26%)	32 (36%)
Others	2 (3%)	1 (5%)	0 (0%)	0 (0%)
Unknown	29 (39%)	1 (5%)	41 (24%)	9 (10%)

ACEI = angiotensin-converting enzyme inhibitor; HIV = human immunodeficiency virus; NSAID = nonsteroidal anti-inflammatory drug.

A total of 137 patients received corrective measures for hyponatremia, 36 (26%) of whom showed a decrease in serum sodium after initiation of treatment during the first 4 days after admission. Reasons for the ongoing decrease of serum sodium were insufficient sodium administration in 9 patients, failed water restriction in 5 patients, and a combination of both in 3 patients. In 2 patients, drugs that were considered being responsible for hyponatremia were stopped without starting other measures. In 17 patients, reasons for the further decrease in serum sodium were not clear from the charts.

The median change in serum sodium during the first 24 hours after admission was 5.0 mmol/L (Q1: 2.0, Q3: 9.0), with a minimum of −4 and a maximum of 26 mmol/L. In 19 patients (13% of all corrected patients), the increase in serum sodium exceeded the maximum recommended correction rate of 12 mmol/L/24 hours. In 3 patients, the correction rate exceeded 20 mmol/L/24 hours. One of the patients with overcorrection was reported to be somnolent after correction; however, hepatic encephalopathy was present during the same time. Syncope was reported in a second patient after correction. A neurologic examination after the syncope revealed no pathologies. For the second and third 24-hour periods, correction rates were 5.0 mmol/L (Q1: 2.9, Q3: 8.0) and 3.0 mmol/L (Q1: 1.0, Q3: 6.0), respectively. Boxplots of the correction rates are shown in **Figure 3**.

Correction rates in patients with and without symptomatic hyponatremia did not differ significantly in the first 24 hours (5.0 vs 6.0 mmol/L, $P = .76$), second 24 hours (6 vs 5 mmol/L, $P = .24$), or third 24 hours (3.0 vs 3.0 mmol/L, $P = .81$). The course of serum sodium in asymptomatic and symptomatic patients is shown in **Figure 4**.

Forty-three patients (26%) were admitted to the intensive care unit directly from the emergency department, 23 of whom presented with symptomatic hyponatremia. Twenty-two patients (13%) died during hospitalization. Causes of death are shown in **Table 3**.

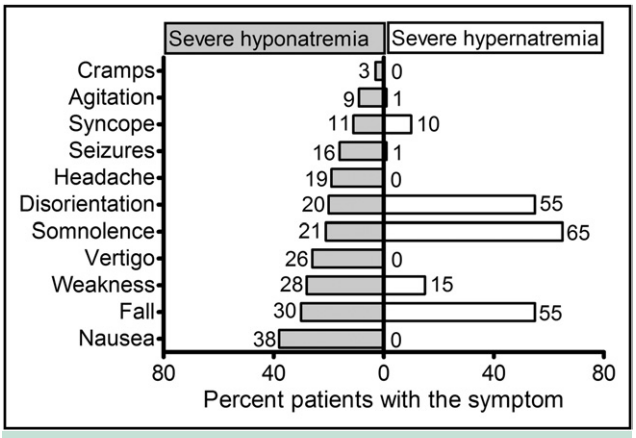

Figure 2 Overview of the neurologic symptoms of hyponatremia and hypernatremia on admission to the emergency department. Numbers are given in percent.

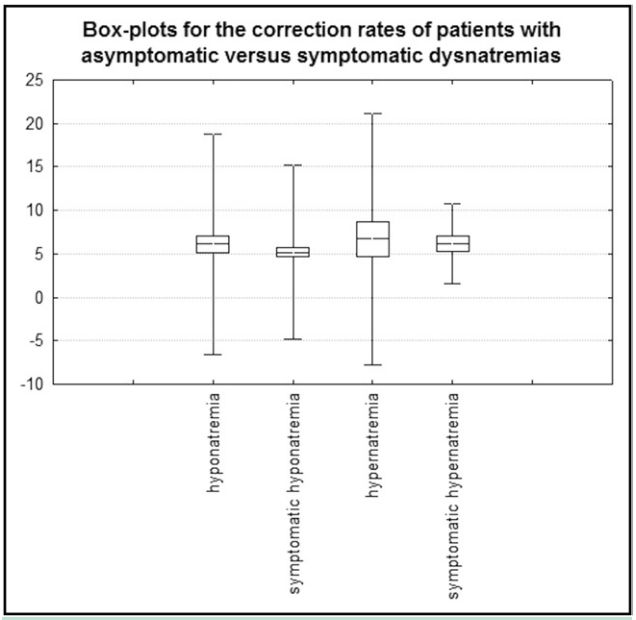

Figure 3 Box-plots for the correction rates of severe hyponatremia and hypernatremia during the first 24 hours after admission to the emergency department. Numbers are in mmol/L/24 hours.

Serum sodium was significantly lower in patients who were admitted to the intensive care unit (115 [Q1: 111, Q3: 117] vs 118 [Q1: 115, Q3: 119] mmol/L, $P < .01$). Serum sodium did not differ in patients who survived and patients who died during hospitalization (116 [Q1: 114, Q3: 119] vs 116 mmol/L [Q1: 115, Q3: 119], $P = .9$).

Severe Hypernatremia

Patient Characteristics. Severe hypernatremia was observed in 74 patients. Forty-six patients (62%) were male. A

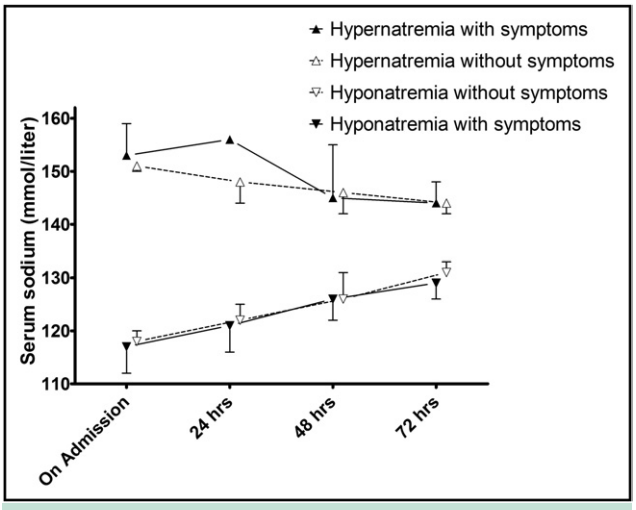

Figure 4 Course of serum sodium for the first 4 days after admission for patients with symptomatic and asymptomatic dysnatremias.

Table 3 Causes of Death for Patients with Severe Hyponatremia and Hypernatremia

Cause of Death (N = 43)	Hypernatremia (N = 21)	Hyponatremia (N = 22)
Pneumonia due to aspiration	2 (5%)	3 (7%)
Sepsis	5 (12%)	2 (5%)
Tumor progression	3 (7%)	3 (7%)
Cerebrovascular ischemia	1 (2%)	0 (0%)
Suicide	1 (2%)	0 (0%)
Esophageal varices bleeding	0 (0%)	1 (2%)
Unknown	9 (21%)	13 (30%)

total of 44 patients (59%) had medical admissions, and 30 patients (41%) had surgical admissions.

Serum sodium on admission of patients with severe hypernatremia was 152 mmol/L (Q1: 150, Q3: 154), with a minimum of 150 mmol/L and a maximum of 177 mmol/L. Serum osmolality was 358 mosmol/kg (Q1: 334, Q3: 378), with a minimum of 293 mosmol/kg and a maximum of 439 mosmol/kg. Serum sodium on admission correlated significantly with serum osmolality ($R = 0.35$, $P < .05$). Creatinine on admission was 102 μmol/L (Q1: 68, Q3: 152), with a maximum of 865 μmol/L. Urine osmolality was 608 mosmol/kg (Q1: 400, Q3: 774) on admission. Three patients (4%) were taking steroids, and 8 patients (11%) were taking loop diuretics. An overview of concomitant medication on admission is shown in **Table 2**.

Symptoms of Severe Hypernatremia. Data on symptoms of hypernatremia were available for 53 of 74 patients. Of these patients, 20 (38%) had neurologic symptoms: Seven patients (35%) had 1 or 2 symptoms; 5 patients (25%) had 3 symptoms; and 1 patient (5%) had 4 symptoms. The most common symptom was somnolence in 13 patients (65%), followed by disorientation/confusion and a recent history of falls in 11 patients (55%) each. **Figure 2** shows an overview of the symptoms. On admission, the median GCS score in patients with severe hypernatremia was 15 (Q1: 11, Q3: 15). The GCS did not correlate with serum sodium levels on admission. Patients with symptomatic hypernatremia had a significantly higher serum sodium level than those who were asymptomatic (153 [Q1: 151, Q3: 159] vs 151 [Q1: 150, Q3: 154] mmol/L, $P = .02$). There was no correlation between the number of symptoms and the serum sodium level ($R = 0.33$, $P > .05$).

Correction of Hypernatremia and Outcome. Data on serum sodium on the day after admission were available for 40 patients (54%). No corrective measures were taken in 13 of 40 patients (18% of all those with severe hypernatremia) during the first 4 days, although 6 of them had documented symptomatic hypernatremia. Corrective measures were taken in 27 patients (36% of all patients with severe hypernatremia). None of them developed a second increase in serum sodium after corrective measures were started. In 7 patients not receiving corrective measures, serum sodium increased further on day 1. Reasons for the further increase were insufficient supply of free water in 4 patients and unknown in 3 patients. In patients with corrective measures, change in serum sodium in the first 24 hours was 6.0 mmol/L (Q1: 3, Q3: 9), with a minimum of 0 and a maximum of 27 mmol/L. The decrease in serum sodium exceeded the recommended correction rate of 12 mmol/L/24 hours or 0.5 mmol/L/h in 3 patients (11%)[1,8] and of 20 mmol/L/24 hours in 1 patient. For the second and third 24-hour periods, correction rates were 4.0 mmol/L (Q1: 1, Q3: 5.5) and 1.0 mmol/L (Q1: 0, Q3: 3). Correction rates in patients with and without symptomatic severe hypernatremia did not differ significantly in the first (6.0 vs 7.0 mmol/L, $P = .62$) or second 24 hours (4.0 vs 3.0 mmol/L, $P = .57$). Box-plots for correction rates during the first 24 hours for symptomatic and asymptomatic patients are shown in **Figure 3**. By including all patients with symptomatic hypernatremia together (those who did and did not receive correction), serum sodium increased from 153 mmol/L on admission to 156 mmol/L after 24 hours.

Seventeen patients (23%) were admitted to the intensive care unit from the emergency department, 3 of whom were symptomatic. There was no difference in serum sodium between patients who were and were not admitted to the intensive care unit (152 [Q1: 152, Q3: 154] vs 151 [Q1: 150, Q3: 155] mmol/L, $P = .5$). Twenty-one patients (28%) died during hospitalization. Causes of death are shown in **Table 3**. Serum sodium did not differ between patients who died and patients who survived (153 [Q1: 150, Q3: 157] vs 152 [Q1: 150, Q3: 154] mmol/L, $P = .27$).

Comparison of Severe Hyponatremia and Severe Hypernatremia

The mean age (60 vs 64 years, $P = .1$) and sex distribution ($P = .4$) in the hyponatremic and hypernatremic groups did not differ. The GCS score on admission was significantly lower in patients with severe hypernatremia than in those with hyponatremia (12 vs 14, $P < .01$). Patients with hypernatremia had higher C-reactive protein levels (65 vs 38 g/L, $P = .02$) and creatinine levels (140 vs 92 μmol/L, $P < .01$). Lactate, bilirubin, hemoglobin, and thyroid-stimulating hormone levels did not differ. Patients with hypernatremia had a significantly higher urine osmolality than patients with hyponatremia (591 vs 363 mosmol/kg, $P < .01$), but urine sodium concentration did not differ (59 vs 46 mmol/L, $P = .2$).

Significantly more patients with hyponatremia presented with symptoms due to the electrolyte disorder (64% vs 38%, $P = .049$). Patients with hyponatremia were more likely to present with weakness (15% vs 4%, $P = .03$), seizures (8% vs 1%, $P = .048$), headache (10% vs 0%, $P = .01$), nausea (20% vs 0%, $P < .01$), and vertigo (14% vs 0%, $P = .02$). There were no significant differences for the other symptoms.

Significantly more hypernatremic patients did not receive correction (18% vs 4%, $P = .02$). Correction rates did not differ for severe hypernatremia and hyponatremia during the first 24 hours after admission (6.0 vs 5.0 mmol/L/24 hours, $P = .42$). Although there was no difference in the number of intensive care unit admissions ($P = .73$), in-hospital mortality was significantly higher in patients with hypernatremia (28% vs 13%, $P = .02$).

No significant differences between men and women were found for symptomatic dysnatremias ($P = .14$) or for the male and female subgroups with hypernatremia ($P = .45$) and hyponatremia ($P = .27$).

DISCUSSION

Dysnatremias were present in 12% of all patients who were admitted to the emergency department. Symptoms were more common in hyponatremic patients than in hypernatremic patients. A significant proportion of patients did not receive corrective measures. Corrective measures were not more rapid in patients with symptomatic dysnatremias. A further increase in serum sodium was even observed in patients with symptomatic hypernatremia in the first 24 hours after admission.

Lee et al[12] reported that 4% of all patients admitted to the emergency department in Taiwan had hyponatremia. Hypernatremia in the population presenting to the emergency department seems not to have been investigated.

The recommended correction rates of 12 mmol/L/d or 0.5 mmol/L/h primarily have been based on pediatric retrospective case series. We investigated correction rates in a broad-based sample. We found that a significant proportion of patients were not treated for dysnatremias. Also, patients with symptomatic dysnatremias did not receive more prompt corrective measures than those who were asymptomatic, although this is recommended widely in the literature for good reasons.[1,8,15-17] In fact, when looking at the absolute numbers, corrective measures were slightly slower in symptomatic patients. This might have been due to a fear that rapid overcorrection of hyponatremia and hypernatremia might have serious adverse effects, such as central pontine myelinolysis or cerebral swelling.[1,8,18]

Serum sodium was not followed in 24 patients with severe hyponatremia and in 34 patients with hypernatremia. Four of 24 patients with severe hyponatremia and 7 of 34 patients with hypernatremia without sodium measurements on the next day died. Four of the patients with hypernatremia and 10 patients with hyponatremia without measured serum sodium on the next day were admitted to the intensive care unit. It cannot be excluded that sodium was measured by local blood gas analyzers.

Hypernatremia has long been considered to be mainly a problem in elderly persons.[19,20] In this study, we showed that patients with hypernatremia were not older than those with hyponatremia. The widespread use of thiazide diuretics has been linked to a higher incidence of hyponatremia.[21-23] Some 14% of our patients were regularly taking thiazide diuretics, and 26% of patients with severe hyponatremia were taking psychotropic drugs, many of which have been associated with the syndrome of inadequate antidiuretic hormone secretion.[24]

In patients with severe hypernatremia, urine osmolality was significantly higher than in patients with hyponatremia, indicating that dehydration plays a major role in its generation.[14,25] Another indicator for this might be that the higher serum creatinine level in patients with severe hypernatremia points to a pre-renal state due to dehydration. Some 11% of patients with severe hypernatremia were taking loop diuretics, which might result in a lower concentration of urine and an increase in serum sodium.[26]

Study Limitations

Our study is limited by its retrospective design. The large numbers of patients presenting to the emergency department provoked the focus on patients with severe dysnatremias because a detailed review of patient charts would be impossible with a more liberal cutoff of serum sodium for inclusion. Symptoms were considered positive if mentioned in the charts only because of the retrospective design of the study. In patients in whom serum sodium measured by the central laboratory for the following days was not available, we cannot exclude that it was measured by local blood gas analyzers. We cannot exclude that patients who did not receive measurement of serum sodium were hyponatremic or hypernatremic.

CONCLUSIONS

Dysnatremias are common in patients presenting to the emergency department. Approximately one third of patients with severe hypernatremia and two thirds of patients with hyponatremia present with neurologic symptoms due to the electrolyte disorder. Symptomatic dysnatremias are not corrected faster than asymptomatic dysnatremias, although clear recommendations exist for this.

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