Hypernatremia at presentation to the emergency department: a case series

Svenja Ravioli1 · Vanessa Rohn1 · Gregor Lindner1,2

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Abstract
Disorders of serum sodium are common findings in patients presenting to the emergency department (ED). The aim of this study was to systematically investigate the prevalence, symptoms, etiology, treatment as well as the course of hypernatremia present on admission to the ED. All adult patients with measurements of serum sodium presenting to the ED between 01 January 2017 and 31 December 2020 were included in this retrospective cohort study. Chart reviews were performed for all patients with hypernatremia defined as serum sodium > 147 mmol/L. 376 patients (0.7%) had a serum sodium > 145 mmol/L on admission and 109 patients (0.2%) had clinically relevant hypernatremia > 147 mmol/L. Main symptoms included somnolence (42%) followed by disorientation (30%) and recent falls (17%). An impaired sense of thirst was the main cause of hypernatremia as present in 76 patients (70%), followed by a lack of free access to water in 50 patients (46%). Regarding treatment, only one patient received targeted oral hydration and 38 patients (35%) experienced inadequate correction of hypernatremia as defined as either a correction of < 2 mmol/L or further increasing sodium during the first 24 h. 25% of patients with hypernatremia died during the course of their hospital stay. Patients who died had significantly lower correction rates of serum sodium (0 mmol/L (−3 − 1.5) versus − 6 mmol/L (−10 − 0), p < 0.001). Hypernatremia is regularly encountered in the ED and patients present with unspecific neurologic symptoms. Initial treatment and correction of hypernatremia are frequently inadequate with no decrease or even increase in serum sodium during the first 24 h.

Keywords Electrolyte · Emergency Hypernatremia · On admission · Sodium

Introduction
Disorders of serum sodium are common in hospitalized patients as well as in patients presenting to the emergency department (ED) [1–3]. Hypo- as well as hypernatremia were identified as predictors for adverse outcome in the critically ill [4, 5]. Furthermore, evidence is growing that sodium disorders are not only markers for disease severity but also impede physiologic functions themselves [6, 7].

In terms of hypernatremia, available data remains scarce. Imai and colleagues found that the prevalence of hypernatremia in elderly patients presenting to the ED was highest in wintertime, suggesting seasonal differences [8]. In a review on dysnatremias in the ED, hypernatremia was predominantly described in the elderly or in patients depending on others such as infants, patients with mental impairment or the critically ill [9]. Severity of hypernatremia mainly depends on the clinical presentation and community-acquired hypernatremia was defined as serum sodium > 147 mmol/L by Jung and colleagues [10].

While hyponatremia has a broad spectrum of etiologies, of which the syndrome of inadequate antidiuretic hormone secretion, low effective circulating volume through heart failure or cirrhosis of the liver or diuretics are amongst the most common [11, 12]. Hypernatremia in outpatients on the other hand, is mainly caused by an inadequate intake of electrolyte-free water, due to a lack of access or an impaired sense of thirst, caused by a central nervous system pathology, medication or intoxication [13, 14]. For hospitalized and critically ill patients the pathophysiology differs relevantly: here, substitution of hypotonic fluid loss by isosmotic or even hyperosmotic fluids is the main cause

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1 Department of Internal and Emergency Medicine, Buergerspital Solothurn, Schoengruenstrasse 42, 4500 Solothurn, Switzerland

2 Department of Emergency Medicine, Inselspital, University Hospital Bern, Bern, Switzerland

Svenja Ravioli
svenja.ravioli@gmail.com
of hypernatremia as was shown by tonicity balance studies [15]. Given these mechanisms leading to hypernatremia, it is not surprising that hypernatremia is considered an indicator of the quality of care in the critically ill, although this most certainly is also true for people in nursing homes or non-critically ill, hospitalized patients [16].

However, despite the fact that hypernatremia is frequently encountered, has serious effects on multiple physiologic functions and represents an independent predictor for an adverse outcome as outlined above, there is a scarcity of studies systematically investigating its etiology, adequacy of treatment as well as the course of the electrolyte disorder.

In the present study, our aim was to evaluate (A) prevalence, (B) symptoms, (C) etiology, (D) treatment as well as (E) outcome of hypernatremia in a series of patients presenting to the emergency department of a large, public hospital with clinically relevant hypernatremia on admission.

Materials and methods
Setting and design
This retrospective cohort analysis of patients with clinically relevant on-admission hypernatremia was conducted at the Department of Internal and Emergency Medicine of the Burgherspital Solothurn, a large, public hospital in central Switzerland. The interdisciplinary ED is the main point of access for patients in need of emergency care 24/7 with approximately 40,000 annual consultations.

Patients selection
All patients admitted to the ED between January 1st 2017 and December 31st 2020 with measurement of serum sodium were eligible for this retrospective analysis. Exclusion criteria were age younger than 18 years or an oral or written withdrawal of consent.

Data collection
Of all patients, data on age, sex, medical history, medication, length of stay and mortality were gathered. Patients were screened for the presence of hypernatremia as defined by serum sodium > 145 mmol/L. To rule out clinically less relevant hypernatremia, the cohort analysis was performed on patients with serum sodium > 147 mmol/L, also described as community-acquired hypernatremia [4, 10]. Chart reviews were performed by two members of the study group (SR, VR) to obtain daily serum sodium levels from day one to five of the hospitalization and at discharge. Furthermore, domicile on admission and destination at discharge were extracted. Etiology of hypernatremia was evaluated on basis of medical history, medication review as well as clinical data such as volume status and vital signs if available. In case of uncertainty concerning the etiology of hypernatremia, the chart was discussed with a senior consultant. Treatment measures were analyzed for the first 24 h after ED admission. Additional outcome measures in patients with hypernatremia were need for intermediate care (IMC) or intensive care (ICU) admission and sodium correction rate.

Statistical analysis
Data were exported to a statistical software package (SPSS for Windows, version 28; SPSS Inc; Chicago, IL) for analysis. Continuous data are presented as median and interquartile ranges or as mean and standard deviation (± SD). Distribution of continuous variables was assessed using normal. Categorical data is presented as absolute counts and percent. Between-group comparisons of continuous variables were performed using Students T test or Mann–Whitney-U tests, respectively. Categorical variables were compared using the χ² test. A two-sided p value < 0.05 was considered statistically significant.

Ethical considerations
This was a retrospective project so no intervention to the patient was conducted. The study was approved by the local ethics committee “Ethikkommission der Nordwest- und Zentralschweiz” (www.EKNZ.ch) and the need for individual informed consent was waived due to the retrospective nature of the study (2021-02186).

Results
During the study period, 53,674 patients with measurements of serum sodium presented to the ED. Mean age of patients overall was 60 years (± 22) and 27,014 (50%) were women. Mean serum sodium was 138 mmol/L (± 3.8), potassium was 3.9 mmol/L (± 0.5) and creatinine was 89 μmol/L (± 60), respectively. Mean length of stay was 4.1 days (± 5.7).

Hypernatremia in the emergency department
In total, 376 patients (0.7%) had a serum sodium exceeding 145 mmol/L and were thus included in the analysis. Maximum serum sodium observed on admission was 175 mmol/L. Mean age of hypernatremic patients was 67 years (± 22) and 178 (47%) were women. Mean length of stay was 5.6 days (± 6.7). Of these, 109 patients (0.2%) had clinically relevant hypernatremia, defined as a serum sodium exceeding 147 mmol/L. Patients with relevant hypernatremia were significantly older than those with mild hypernatremia.
(72 years (± 22) versus 67 (± 22), \( p = 0.002 \)). There was no difference in the distribution of sex between relevant and mild hypernatremia (50% versus 46%, \( p = 0.6 \)), but patients with relevant hypernatremia had a significantly longer length of stay (7.4 (± 6.7) versus 4.9 (± 6.2), \( p < 0.001 \)).

**Characteristics of patients with clinically relevant hypernatremia**

Of all patients with hypernatremia > 147 mmol/L (i.e., 109), 47 patients (43%) were referred to the ED from home, while 51 (47%) were referred from a nursing home and 11 (10%) from a different hospital including psychiatry wards or rehabilitation institutions. The main reasons for ED presentation in hypernatremic patients were infection (44%), mainly sepsis (18%) or pneumonia (16%), trauma (9%) and intoxication (9%). 4 out of 109 patients (4%) were tested positive for COVID-19 on admission. Hypernatremia was also present in patients referred to the ED with neurologic symptoms including seizures (9%), gastrointestinal problems (7%) and respiratory problems including pulmonary embolism (6%). In 23 patients (21%), no symptoms typically attributable to hypernatremia were present on ED admission as identified by chart review. Somnolence was the most common symptom, present in 46 hypernatremic patients (42%), followed by disorientation 33 (30%), history of fall in 18 (17%) and severe fatigue in 11 patients (10%), respectively. A detailed overview of symptoms attributable to hypernatremia is given in Fig. 1. In total, 70 patients (64%) were clearly described to be hypovolemic on admission by the ED physicians in charge. In 36 patients (33%), diuretic medication was present on admission to the ED of which 30 (28%) were loop diuretics.

**Etiology of hypernatremia**

Concerning the etiology of hypernatremia, 76 patients (70%) suffered from an impaired sense of thirst whereas 50 patients (46%) had no access to free water due to immobility for example. Renal loss of free water caused by diuretics was present in 35 patients (32%) and 14 patients (13%) had increased gastrointestinal fluid loss. 11 patients (12%) had other identifiable causes for hypernatremia, such as severe dehydration due to massive blood loss, intoxication with lithium or impossibility to swallow due to bolus impaction.

**Treatment of hypernatremia**

In the ED, only one patient explicitly received targeted oral hydration as a treatment for hypernatremia. 98 patients (90%) received intravenous hydration: 53 patients (49%) received a balanced crystalloid solution, 9 patients (8%) solely received a glucose 5% solution and 2 patients (2%) received a 0.9% saline solution. 31 patients (28%) received a combination of a balanced crystalloid and 5% glucose and 3 patients (3%) had a combination of a balanced crystalloid and 0.9% saline. 11 patients (10%) did not receive hydration at all. Median total fluid volume infused during the first 24 h after admission was 1500 ml (800–2500).

**Course and outcome of hypernatremia**

In total, 38 patients (35%) were considered inadequately corrected during the first 24 h after admission: in 20 patients (18%) a further rise of serum sodium was detected, while 8 patients (7%) had no decline of serum sodium and 7 patients (6%) had a decline of less than 2 mmol/L/24 h. In 3 patients (3%) an overcorrection of

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**Fig. 1** Symptoms of hypernatremia on admission to the emergency department

![Symptoms of Hypernatremia on Admission](chart)
serum sodium of > 10 mmol/L was measured. The course of serum sodium over the first 5 days after hospitalization is depicted in Fig. 2.

3 patients (3%) were admitted to the intermediate care unit and 17 patients (16%) to the intensive care unit. 27 patients (25%) with hypernatremia died during hospitalization. Serum sodium on admission was not different between the patients that survived and the deceased (150 mmol/L (148–152) versus (151 mmol/L (148–156), p = 0.32). Delta serum sodium between admission and discharge/death was significantly higher in patients who survived (-6 mmol/L (-10-0) versus 0 mmol/L (-3-1.5), p < 0.001). Table 1 gives an overview of patients who survived compared to those who died.

Of the 82 patients discharged from the hospital, 33 patients (40%) were still hypernatremic at the time of discharge. Of the 37 surviving patients being admitted from home, 7 (19%) were newly discharged to a nursing home and 9 (24%) were discharged to another hospital including rehabilitation or psychiatry. All 34 survivors admitted from a nursing home were discharged to the same destination.

**Discussion**

The present study showed that on-admission hypernatremia is by far less common than hyponatremia but still frequently observed in the ED. It was shown that the disorder is not

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**Table 1** Comparison of course and outcome of patients with clinically relevant hypernatremia

|                        | Deceased                  | Survivors                  | p value |
|------------------------|---------------------------|----------------------------|---------|
| N                      | 27 (25%)                  | 82 (75%)                   | –       |
| Median serum sodium (mmol/L) | 151 (148–156)          | 150 (148–152)              | 0.32    |
| Median delta sodium in 24 h (mmol/L) | – 1 (– 2.25 – 2.25)  | – 2 (– 4 – 1)              | 0.19    |
| Median delta sodium at discharge (mmol/L) | 0 (– 3 – 1.5)          | – 6 (– 10 – 0)             | <0.001  |
| Median length of hospital stay (days) | 5 (2–7)                   | 6 (2–10)                   | 0.2     |
exclusive to the very elderly population living in nursing homes as can be seen by the mean age of 67 years of hyponatremic patients and almost half of them being referred from home. Most common reasons for ED referral in patients with clinically relevant hyponatremia were infections, mainly sepsis and pneumonia, followed by trauma and intoxications. Furthermore, it was found that hypernatremia treatment was inadequate in many cases with serum sodium either remaining constant or even increasing during the first 24 h after ED admission. Mortality in hypernatremia > 147 mmol/L was high at 25%, while patients with adverse outcome had significantly less decline in serum sodium than those who survived.

The results of this study including the prevalence of hypernatremia defined as serum sodium of > 145 mmol/L of 0.7% and the prevalence of clinically relevant on-admission hypernatremia defined as > 147 mmol/L of 0.2% are consistent with previous findings [2, 8]. No statistically significant rise in hypernatremia prevalence was found during the COVID-19 pandemic as could have been suspected due to staff shortages in nursing homes and consequent deterioration in the quality of care of inhabitants (data not shown).

The main symptoms attributable to hypernatremia were somnolence, disorientation as well as falls. These findings stand in line with the limited evidence available [2]. These symptoms can be well explained by the effect of hypernatremia and its associated hyperosmolality on the central nervous system [13]: extracellular hyperosmolality leads to a shift of free water from the intra- to the extracellular space and consequent cerebral dehydration, which is counteracted over the longer term.

Although several reviews discussed the mechanisms leading to hypernatremia, there is a lack of systematic investigations of the etiology of on-admission hypernatremia. In the present study, an impaired sense of thirst paired with limited access to free water was the main reason for the development of hypernatremia, often as a combination of both. However, increased fluid loss caused by diuretics was also an identifiable cause in one of three patients. Very similar to critically ill patients, these results imply that hypernatremia and its prevalence can be considered an indicator of neglect in persons living in institutions such as nursing homes [16].

One of the most intriguing findings of the present study is the commonly encountered insufficient treatment of hypernatremia: as many as 35% of patients experienced stable or even rising serum sodium levels during the first 24 h after admission to the ED. This might be caused on the one hand by a lack of awareness of hypernatremia itself or underestimating its adverse effects. On the other hand, an inadequate choice of fluid therapy might be an explanation for our findings: hypotonic fluids were part of the initial hydration therapy in only a third of the patients while targeted oral hydration was used in one single patient only. Strikingly, many patients had only small or lacking corrections of serum sodium during the entire course of hospitalization. On the other end of the spectrum, three patients (3%) were even found to be overcorrected with a decline in serum sodium exceeding 10 mmol/L during the first 24 h after admission. These results underline that the creation of awareness for hypernatremia as well as its adverse effects on patients must be enforced, especially in physicians working in internal and emergency medicine.

The finding that deceased patients had a significantly lower reduction of serum sodium than those who survived could be interpreted as insufficient treatment, but may as well be an expression of a more severe underlying disease. This question cannot be answered on basis of the present analysis.

The present study is limited by its retrospective design. Therefore, insufficient documentation cannot be excluded in some cases. Moreover, we were dependent on detailed documentation as well as the correctness of the clinical evaluation of the physicians in charge of the included patients.

In conclusion, we could find that relevant on-admission hypernatremia is encountered on a low but regular basis in the ED, mainly caused by an impaired sense of thirst and/or lack of access to free water. Despite the measurement of serum sodium hypernatremia appears to be frequently neglected and treatment was often inadequate during the first 24 h after admission.

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Declarations

Conflict of interest The authors declare that they have no conflict of interest.

Human and animal rights statement and Informed consent This was a retrospective study and no intervention to the patient was conducted. The study was approved by the local ethics committee “Ethikkommission der Nordwest- und Zentralschweiz” and the need for individual informed consent was waived due to the retrospective nature of the study (2021-02186).

References

1. Arampatzis S, Funk GC, Leichtle AB, Fiedler GM, Schwarz C, Zimmermann H, Exadaktylos AK, Lindner G (2013) Impact of diuretic therapy-associated electrolyte disorders present on admission to the emergency department: a cross-sectional analysis. BMC Med 27(11):83. https://doi.org/10.1186/1741-7015-11-83
2. Arampatzis S, Frauchiger B, Fiedler GM, Leichtle AB, Buhl D, Schwarz C, Funk GC, Zimmermann H, Exadaktylos AK, Lindner G (2012) Characteristics, symptoms, and outcome of severe dysnatremias present on hospital admission. Am J Med 125(11):1125–1129.e1-1125.e7
3. Lindner G, Exadaktylos AK (2013) Disorders of serum sodium in emergency patients: salt in the soup of emergency medicine. Anaesthesist 62(4):296–303

4. Funk GC, Lindner G, Druml W, Metnitz B, Schwarz C, Bauer P, Metnitz PG (2010) Incidence and prognosis of dysnatremias present on ICU admission. Intensive Care Med 36(2):304–311

5. Lindner G, Funk GC, Schwarz C, Kneidinger N, Kaider A, Schneeweiss B, Kramer L, Druml W (2007) Hypernatremia in the critically ill is an independent risk factor for mortality. Am J Kidney Dis 50(6):952–957

6. Lenz K, Gössinger H, Laggner A, Druml W, Grimm G, Schneeweiss B (1986) Influence of hypernatremic-hyperosmolar state on hemodynamics of patients with normal and depressed myocardial function. Crit Care Med 14(10):913–914

7. Josiassen RC, Filmyer DM, Geboy AG, Martin DM, Curtis JL, Shaughnessy RA, Salzman A, Orlandi C (2012) Psychomotor deficits associated with hyponatremia: a retrospective analysis. Clin Neuropsychol 26(1):74–87

8. Imai N, Sumi H, Shibagaki Y (2019) Impact of age on the seasonal prevalence of hypernatremia in the emergency department a single-center study. Int J Emerg Med 12(1):29

9. Pfennig CL, Slovis CM (2012) Sodium disorders in the emergency department: a review of hyponatremia and hypernatremia. Emerg Med Pract 14(10):1–26

10. Jung WJ, Lee HJ, Park S, Lee SN, Kang HR, Jeon JS, Noh H, Han DC, Kwon SH (2017) Severity of community acquired hypernatremia is an independent predictor of mortality. Intern Emerg Med 12(7):935–940

11. Burst V (2019) Etiology and epidemiology of hyponatremia. Front Horm Res 52:24–35. https://doi.org/10.1159/000493234

12. Ravioli S, Bahmad S, Funk GC, Schwarz C, Exadaktylos A, Lindner G (2021) Risk of electrolyte disorders, syncope, and falls in patients taking thiazide diuretics: results of a cross-sectional study. Am J Med 134(9):1148–1154. https://doi.org/10.1016/j.amjmed.2021.04.007

13. Adrogue HJ, Madias NE (2000) Hypernatremia. N Engl J Med 342(20):1493–1499. https://doi.org/10.1056/NEJM2000051834206

14. Agrawal V, Agarwal M, Joshi SR, Ghosh AK (2008) Hyponatremia and hypernatremia: disorders of water balance. J Assoc Physicians India 56:956–964

15. Lindner G, Kneidinger N, Holzinger U, Druml W, Schwarz C (2009) Tonicity balance in patients with hypernatremia acquired in the intensive care unit. Am J Kidney Dis 54(4):674–679. https://doi.org/10.1053/j.ajkd.2009.04.015

16. Polderman KH, Schreuder WO, van Strack Schijndel RJ, Thijs LG (1999) Hypernatremia in the intensive care unit: an indicator of quality of care? Crit Care Med 27(6):1105–1108. https://doi.org/10.1097/00003246-199906000-00029

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