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A Comparison of Community-Acquired and Hospital-Acquired Hypernatraemia in Patients who are Acutely Admitted to Hospital

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Abstract

Background

Hypernatraemia is associated with a short-term mortality of 20-60%. Age-related physiological changes predispose patients to hypernatraemia. This study reviewed acutely admitted patients comparing those with community-acquired (CAH) and hospital-acquired hypernatraemia (HAH).

Methods

A retrospective study of 102 consecutive acute medical in-patients with serum [Na]>145 mmol/L was conducted. Baseline characteristics, clinical presentation, laboratory values, monitoring, management and outcomes were compared between CAH and HAH groups.

Results

Patients were exclusively older (>69 years). Forty patients (39.2%) had CAH and sixty-two (61.8%) had HAH. Those with CAH were more likely to be NH residents, have dementia and reduced mobility. Most HAH patients had mild hypernatraemia initially (75.8%, n=47), and higher rates of acute kidney injury (27% (n=11) vs 8% (n=3)/p=0.02) were observed. Monitoring was inadequate and no patient had a free water deficit documented. Medication review and intravenous fluid prescribing was similar between groups. The median length of stay of discharged HAH patients was longer (22.5 vs 8 days/p=0.005). Mortality rates were similar (47% (n=29) vs 37% (n=15)/p=0.416). Time from admission to death was higher in HAH patients (16 vs 8 days/p=0.008).

Conclusions

Both CAH and HAH present similarly, however, older patients with cognitive/physical impairments are at an increased risk. Early identification of high-risk patients and adherence to best practice guidelines is required.

Introduction

Hypernatraemia is associated with significant morbidity and a short-term mortality between 20-60%[1-3]. Hospitalised patients may present with or acquire hypernatraemia during their admission. Age-related decline in organ function, appetite, illness and disability, and increased fluid requirements predispose older adults to dehydration and hypertonicity[4-6]. Thirst is the main line of defence against hypernatraemia. Patients with an intact thirst mechanism can sense a rise in serum osmolality and rectify this by sourcing and consuming water[7]. Impaired thirst occurs with normal ageing[8]. Additionally, cognitive and physical disabilities act as barriers to this process, predisposing to dehydration[9].

Community-acquired hypernatraemia (CAH) is present in 1-2% of Emergency Department admissions and has been less well studied than hospital-acquired hypernatraemia (HAH) [10]. CAH is usually hypovolaemic, and associated with a lower mortality compared with HAH[11,12]. One study on CAH found the presence of Alzheimer's disease, impaired oral intake and concomitant treatment with Renin Angiotensin-System (RAS) blockers were positively associated with the development of hypernatraemia[13].

Hypernatraemia may be acquired during hospitalisation, especially in severely unwell patients due to the combination of being unable to drink sufficient water; poor urine concentrating ability due to renal failure; osmotic diuresis from high serum urea concentrations, and water losses through large urine or gastrointestinal outputs[14,15]. HAH is an independent mortality risk factor both in critical care and non-critical care environments[16-18]. It has a worse prognosis than CAH and patients tend to do worse than those with other electrolyte abnormalities such as hyponatraemia[19] The relative contribution that hypernatraemia adds to the poor prognosis in critically unwell patients is unclear. One study found hypernatraemia directly contributed to mortality in 16% of cases[1].

Hypernatraemia requires preventative measures and active management on the part of the treating physician. Every patient should undergo a thorough evaluation of underlying causes, calculation of free-water deficit, replacement via oral/intravenous routes. Serum sodium levels should be monitored to ensure the rate of correction is appropriate. The risks of over-correction appear to be negligible however, and it is now recognised that under-correction poses the greater risk as longer duration of hypernatraemia is associated with poorer outcomes than the absolute sodium levels reached[20]. Frequent monitoring should be performed as per best practice guidelines[21-23]. The authors have recently submitted a description of suboptimal management and monitoring of this patient population[24].

The authors hypothesized that patients with hypernatraemia on presentation and those that acquire it may differ in key patient characteristics, time course of their illness and approach to their management. The aim of this study was to perform a descriptive review and compare community-acquired and hospital-acquired hypernatraemia in terms of patient demographics, clinical presentation, co-morbidities, changes in sodium and renal function, management, and patient outcomes.

Methods

Ethical Approval was granted from the Research Ethics Committee of National University of Ireland, Galway. A retrospective cross-sectional study was conducted. The study population was defined as general medical in-patients with serum sodium concentrations >145 mmol/L using the Galway University Hospital Laboratory Information System (GUH-LIS). A sample of 145 patients was chosen using anonymised medical record numbers. Inclusion criteria were age >18 years, medical admission for >24 hours and availability of electronic or paper-based medical notes. Exclusion criteria were direct ICU admissions, admissions under oncology/haematology/surgical specialities, death/discharge <24 hours of admission and duplicate samples.

The following data was collected retrospectively: age, sex, clinical presentation, co-morbidities, and medications. The GUH-LIS was interrogated to obtain serum biochemistry parameters performed using Roche Diagnostics Cobas® 8000 chemistry analyser.

Evaluation of the management was based on calculated free water deficit, frequency of biochemical monitoring, medication review and intravenous fluid prescriptions. Primary diagnosis and patient outcomes were retrieved from electronic discharge summaries. All collated data was recorded in Microsoft Excel® 2016 and statistical analysis performed using Minitab® 2018. Parametric data were represented as mean (standard deviation) and compared using student's independent t-test. Non-Gaussian data was represented as median (interquartile range) and compared using Kruskal-Wallis test. Comparison of proportions was performed using the chi-squared test. A p-value <0.05 was deemed statistically significant.

Results

Baseline Characteristics

There were 102 patients included, 63% were male. The baseline characteristics are detailed in Table 1 below. Forty patients had CAH and sixty-two had HAH. More HAH patients were admitted from home (68% vs 30%/p=<0.001) and CAH patients included more NH residents (55% vs 32%/p=0.026). A small number of CAH group were admitted from other care pathways. The median age of both groups was similar (81(73.8-87.3) vs 80(69.3-87.8) years/p=0.5). The clinical presentation of admitted patients were categorised as presenting with features of hypovolaemia, reduced level of consciousness (LOC) or infection. Patients could be assigned to more than one category, if applicable. There was no difference in frequency of these presentations, however, more CAH patients presented with all three features (35% vs 15%, p=0.027). CAH patients had higher frequency of dementia (75% vs 44%/p=0.004) and reduced mobility (70% vs 48%/p=0.05). The frequency of diabetes, CKD and cardiovascular disease was similar. Those prescribed regular diuretics (44% vs 28%, p=0.154) and RAS blockers (27% vs 10%, p=0.061) were similar.

Laboratory Results

The mean Sodium concentration ([Na]) on admission was higher in CAH patients (153.6 (+/-6.14) vs 138.7 (+/-4.6)/p<0.001). In HAH patients, 79% (n= 49) had normal [Na], and 21% (n=13) low [Na] on presentation. More HAH patients had mild hypernatraemia (75.8% vs 35%/p<0.001) on the first hypernatraemic sample. More CAH patients had moderate (5% vs 16.1%/p=0.034) and severe (27.5% vs 8,1%/p=0.012) hypernatraemia on initial sampling. The median time from admission to HAH was 4 (2-10.25) days. CAH patients had a higher maximum recorded Sodium ([Na]) recorded, 156(153-160.75) vs 153(151-157), p=0.002. The proportion of patients with a [Na]>160 mmol/L was also higher in CAH group, 28% vs 8%, p=0.008.

The median [Urea] on admission was higher in CAH group, 15.3(8.8-22.3) vs 8.95(6.3-12.9), p=0.002. There was no difference in the median [Creatinine] on admission, 106.5(70.5-197.8) vs 100.5(77.5-138.3), p=0.437.

There was no difference between median [Urea] or [Creatinine] on admission in CAH group compared to the renal profiles of the HAH group at the time of developing hypernatraemia. More HAH patients had an acute kidney injury (AKI) during their admission (27% vs 8%/p=0.02). There was no difference in frequency of AKI on presentation (29% vs 45%/p=0.137).

***Table 1**: Baseline characteristics of study population.*

Study Characteristic	HAH% (n=62)	CAH % (n=40)	P value
Males	63% (39)	63% (25)	1.000
Age^	81 (73.8-87.3)	80 (69.3-87.8)	0.537
Source of Patient			
Nursing Home	32.3% (20)	55% (22)	**0.026**
Home	67.7% (42)	30% (12)	**<0.001**
Other Hospital	0	7..5% (3)	0.058
Psychiatric Hospital	0	5% (2)	0.151
Other residential facility	0	2.5% (1)	0.392
Clinical Presentation			
Hypovolaemia	74% (46)	83% (7)	0.461
Reduced LOC	55% (34)	68% (27)	0.286
Infection	63% (39)	75% (30)	0.290
All three	15% (9)	35% (14)	**0.027**
Co-morbidities			
Dementia	44% (27)	75% (30)	**0.004**
Reduced Mobility	50% (30)	70% (28)	**0.052**
Diabetes Mellitus	11% (7)	20% (8)	0.354
CKD	18% (11)	178% (7)	1.000
Cardiovascular Disease	82% (51)	70% (28)	0.229

Regular Medications			
Diuretics	44% (27)	28% (11)	0.154
RAS Blockers	27% (17)	10% (4)	0.061
Admission Laboratory Values: Sodium/[Na] mmol/L,[Urea] mmol/L, [Creatinine] umol/L % (n)			
Serum sodium/[Na]*	138.7 ((± 4.6)	153.6 ((± 6.14)	<0.001
Serum sodium/[Na]^	139 (135.75-142)	153 (149-157.5)	<0.001
Serum Urea^	8.95 (6.27-12.90	15.3 (8.83-22.27)	**0.002**
Serum Creatinine^	100.5 (77.5-138.3)	106.5 (70.5-197.8)	0.437
Severity of hypernatraemia on first sample: [Na] mmol/L: % (n)			
Mild 146-150	75.8% (47)	35% (14)	**<0.001**
Moderate 151-155	16.1% (10)	35% (14)	**0.034**
Severe > 156	8.1% (5)	27.5% (11)	**0.012**
Renal Profile on initial hypernatraemic sample: % (n)			
Serum Urea^	12.65 (8.43-17.85)	15.3 (8.83-22.27)	0.165
Serum Creatinine^	97 (69.5-165.5)	106.5 (70.5-197.8)	0.399
Highest Sodium Recorded: % (n)			
Highest recorded*	154 ((±3.6)	157.9 ((± 6.9)	**0.002**
Highest recorded^	153 (151-157)	156 (153-160.75)	**0.002**
Mild (145-149 mmol/L)	5% (3)	0	0.278
Moderate (150-154 mmol/L)	53% (33)	35% (14)	0.103
Severe (155-159 mmol/L)	34% (21)	38% (15)	0.832
Very Severe >160 mmol/L)	8% (5)	28% (11)	**0.008**
Acute Kidney Infection (AKI): % (n)			
On admission	29% (18)	45% (18)	0.137
During hospitalisation	27% (17)	8% (3)	**0.020**
No AKI	44% (27)	48% (19)	0.839

*CAH: community acquired hypernatraemia. CKD: Chronic Kidney Disease HAH: hospital acquired hypernatraemia LOC: level of consciousness, RAS: renin angiotensin system, *: data stated as mean ± standard deviation, ^: data stated as median (Interquartile range)*

Management of Hypernatraemia

Monitoring of hypernatraemic patients was suboptimal. More HAH patients did not have a [Na] measured at 12 hours (90% vs 70%/p=0.015). There was no difference between monitoring at 24-hours, (19% vs 35%/p=0.103) or 48-hours (13% vs 5%/p=0.308). More CAH patients had q-12 hourly [Na] measurements over the initial 48 hours period of hypernatraemia (15% vs 3%/p=0.054). No evidence of a calculated free water deficit was found for any patient.

In HAH patients, of those prescribed diuretics, 55% (n=15) had no change, 30% (n=8) were stopped/held and 15% (n=4) had a dose reduction. In the CAH group, 55% (n=6) had no change and 45% (n=5) were stopped/ held. For those prescribed RAS blockers, 76% (n=13) of HAH group and 75% (n=3) of CAH group continued these medications.

Hypotonic fluids (5% Dextrose or 0.45% NACL) were prescribed in 32% (n=20) of HAH and 42.5% (n=17) of CAH group. Volume resuscitation with 0.9% NACL followed by hypotonic fluids was prescribed in 4.8% (n=3) of HAH and 10% (n=4) of CAH group. Isotonic fluids (0.9% NACL or Hartman's solution) were prescribed in 50% (n=31) of HAH and 40% (n=16) of CAH group. No fluids were prescribed in 4.8% (n=3) of HAH and 5% (n=2) of CAH group, see *Table 2* below.

Table 2: *Management of hypernatraemia: monitoring, medication review and intravenous fluid regime.*

Management	HAH (n=62)	CAH (n=40)	P value
Monitoring of Serum [Na]			
No monitoring at 12 hours	90% (56)	70% (28)	0.015
No monitoring at 24 hours	19% (12)	35% (14)	0.103
No monitoring at 48 hours	13% (8)	5% (2)	0.308
Q12 hour monitoring for 48 hours	3% (2)	15% (6)	**0.054**
Medications: % (n)			
Diuretics	HAH (n=27)	CAH (n=11)	
Stopped/Held	30% (8)	45% (5)	0.457
No change	55% (15)	55% (6)	1.000
Reduced	15% (4)	0	0.303
ARB/ACEi	HAH (N=17)	CAH (N=4)	
Stopped/Held	24% (4)	25% (1)	1.000
No change	76% (13)	75% (3)	1.000
Calculation of Free Water Deficit: (n)			
Documented Calculation of Free Water Deficit	0% (0)	0% (0)	1.000
Intravenous Fluid Regimes: (n)			
Hypotonic Fluids	32% (20)	42.5% (17)	0.302
Volume resuscitation followed by hypotonic fluids	4.8% (3)	10% (4)	0.426
Isotonic/Hypertonic Fluids	50% (31)	40% (16)	0.416
No fluids/Dialysis	4.8% (3)	5% (2)	1.000
Missing Data	8% (5)	2.5% (1)	0.399

CAH: Community-acquired Hypernatraemia. HAH: Hospital acquired Hypernatraemia, ^: data stated as median (Interquartile range), LOC: level of consciousness, ARB: Angiotensin Receptor Blocker, ACEi: Angiotensin Converting Enzyme inhibitor; 0.9% NaCl: Normal Saline, P-value ≤0.05 deemed statistically significant.

Outcomes

Hypernatraemia resolved in 45% (n=28) HAH and 35% (n=14) CAH patients, p=0.410. The median duration was similar between groups (5 (2-8.5) vs 4 (2.75-8)/p=0.906). There was no difference in ICU admissions, 13% (n=8) vs 2.5% (n=1), p=0.085.

The most common primary diagnosis was LRTI in both groups 56% (n=35) in HAH and 63% (n=25) in CAH. Infection from other sources, most commonly urinary, was the next most common diagnosis 23% (n=14) and 20% (n=5) respectively, Acute neurological events, including stroke, traumatic brain injury and seizures accounted for 20% (n=14) of HAH and 7.5% (n=3) of CAH group. Decompensated heart failure accounted for 11% (n=7) of HAH and 2.5% (n=1) CAH group.

There was no significant difference in the discharge rate or destination between groups. The discharge rate of HAH patients was 53% (n=33) and 63% (n=25) in CAH patients. In HAH group, 33% (n=11) were discharged home, 39% (n=13) to previous NH, 12% (n=4) were new NH discharges. Other discharge destinations included hospice, 6% (n=2) and rehabilitation, 9% (n=3). In CAH group, 64% (n=16) were discharged to previous NH, 20% (n=5) to home, and 16% (n=4) to hospice care. The median length of stay (LOS) for HAH group was significantly longer than for CAH, 22.5 (9-48.75) vs 8 (5-17) days, p=0.005.

The mean sodium on discharge was similar, 142.1.8 (± 5.7) vs 144.5 (± 6.7) mmol/L, p=0.144. Hypernatraemia was persistent on day of discharge in 30% (n=10) of HAH and 36% (n=9) CAH group. More CAH patients had hypernatraemia documented on discharge correspondence, 18% (n=6) vs 52% (n=13), p=0.011.

The mortality rate was similar between groups, 47% (n=29) vs 37% (n=15), p=0.416. The median time from admission to death was longer in the HAH group, 16 (10.25-22.50) vs 8 (2-14) days, p=0.008. See *Table 3* below

Table 3: *Outcomes of CAH and HAH patients.*

Study Cohort	HAH (n=62)	CAH (n=40)	P Value
Resolution: % (n)			
Full Resolution	45% (28)	35% (14)	0.410
Duration of Hypernatraemia: Serum [Na]>145mmol/L			
Duration/days*	6.4 (± 5.3)	6 (±5.0)	0.800
Duration/days^	5 (2-8.5)	4 (2.75-8)	0.906
Diagnosis			
LRTI	56% (35)	63% (25)	0.681
Other infection	23% (14)	20% (5)	0.298
Stroke/TBI	15% (11)	5% (2)	0.073
Seizures	5% (3)	2.5% (1)	1.000
ADHF	11% (7)	2.5% (1)	0.144
Other	11% (7)	18% (7)	9.392

Critical Care			
ICU Admission	13% (8)	2.5% (1)	0.085
Discharges			
Patients Discharged	53% (33)	63% (25)	0.416
Discharge Destination			
Home	33% (11)	20% (5)	0.375
Previous NH	39% (13)	64%(16)	0.111
New NH	12% (4)	0% (0)	0.126
Hospice	6% (2)	16% (4)	0.387
Rehab/Convalescence	9% (3)	0% (0)	0.251
Serum sodium concentration at Discharge (DC)			
Sodium* (mmol/L)	142.1.8 (± 5.7)	144.5 (±6.7)	0.144
Hypernatraemia on DC	30% (10)	36% (9)	0.779
Hypernatraemia on DC letter	18% (6)	53% (13)	**0.011**
Length of Stay (LOS)			
Number of days^	22.5 (9-48.75)	8 (5-17)	**0.005**
Mortality: % (n)			
Number of Deaths	47% (29)	37% (15)	0.416
Median no. of days to death	16 (10.25-22.50)	8 (2-14)	**0.008**

*NH: Nursing Home, *: data stated as mean ± standard deviation, ^: data stated as median (Interquartile range), ICU: Intensive Care Unit. NH: Nursing Home, LRTI: Lower respiratory tract infection, TBI: Traumatic brain injury, DC: Discharge ADHF: Acute decompensated heart failure, P-value ≤0.05 deemed statistically significant.*

Discussion

This retrospective review of revealed key differences between hospital-acquired and community-acquired hypernatraemia. Although clinical presentations and primary diagnoses, were similar, they differed in their illness trajectory.

Those with CAH were more likely to be NH residents with dementia and reduced mobility. Previous studies reported 10-fold higher prevalence and 2-fold risk of in-hospital mortality in NH residents[25]. Hypernatraemia in CAH was more severe and reached higher maximum values, and thus a much larger free water deficit. This possibly reflects multi-morbidity and reduced access to laboratory testing in some community settings.

HAH developed within days of hospitalisation and progressed in hospital to a greater extent than CAH. This may represent either a failure to recognise or treat hypernatraemia appropriately in the early stages, or the development of hypernatraemia in the setting of progressive severe illness.

Overall monitoring of hypernatraemia in the entire population was suboptimal as highlighted in a previous article[25]. Here, the CAH patients had earlier and more frequent monitoring, potentially due to the severity of their hypernatraemia at diagnosis. Many patients continued to be prescribed potentially inappropriate medications.

Less than one-third of HAH patients and less than half of CAH patients received hypotonic fluids, the preferred choice for correction in the absence of haemodynamic compromise. Under-correction was universal, and no patient was over-corrected.

The most common diagnosis was LRTI, a similar finding to other published studies[19], while other infections and acute neurological events accounted for most other diagnoses in keeping with the admitting presentations. LOS of those discharged was longer in those with HAH, possibly reflecting hypernatraemia occurring in tandem with an evolving prolonged and severe illness and hence a longer hospital stay[1]. Although mortality rates were similar, time from admission to death was longer in the HAH group, likely reflecting a higher proportion of independently living adults with a higher functional baseline. Conversely the higher number of NH residents with cognitive and physical impairment along with more severe hypernatraemia, reflected severe frailty in the CAH group which is associated with earlier recognition of irreversible pathology and decisions regarding ceilings of care[10].

Strengths of this study include a representative real-world study sample and thorough data collection using multiple sources. Limitations include retrospective design, single-centre study and a relatively small sample size. Hypernatraemia should be considered as a marker of quality of care. Earlier identification (especially in HAH) and addressing provision of adequate fluid intake early in their course could result in quicker resolution of hypernatraemia which is associated with better outcomes. A quality improvement initiative aimed at adherence to best practice guidelines for management of both CAH and HAH is planned for our hospital on the basis of these findings.

Ethical approval:
Ethical approval was granted from the Research Ethical Committee of National University of Ireland, Galway.

Declaration of Conflicts of Interest:
The authors declared no potential conflict of interest that could be perceived as prejudicing the impartiality of the research, authorship, and/or publication of this article. The authors received no financial support for the research, authorship and/or publication of this article.

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