Original Article

Describing Outcomes of Hyponatremic Patients Based on the Location Where Urine Chemistry Was First Performed

San Chyi Boon, Nik Azlan Nik Muhamad.

Department of Emergency Medicine, Universiti Kebangsaan Malaysia Medical Centre (UKMMC)
Jalan Yaacob Latif, Bandar Tun Razak, 56000 Kuala Lumpur, Wilayah Persekutuan, MALAYSIA

Abstract

We aimed to study the outcomes of hyponatremic patients on the basis of the initial urine chemistry location (emergency department [ED] vs. inpatient care ward) in this prospective observational cohort study. The population studied was adult patients (>18 years old) with hyponatremia (serum sodium<136 mEq/L). The outcomes compared between the two groups were hospital length of stay (LOS), 24-hour sodium correction rates, and modifications in management following urine osmolarity and sodium results. Among the 231 patients studied, 58 (25.1%) were tested for urine chemistry in the ED, and the remaining 173 (74.9%) were tested in the ward. The median LOS in the group of patients where tests were performed in the ED was 6 days (n = 58) versus 5 days (n = 173), U = 4107.0, z = -2.08, p = 0.038, r = 0.14. Although the LOS difference was statistically significant, the small effect size (r=0.14) indicates limited clinical significance. The median sodium correction rates were significantly greater in the ED group (0.27 mEq/L/hour vs. 0.1 mEq/L/hour; U=3225.0, z=-4.08, p<0.001, r=0.27). Comparisons for modification of management in the first 24 hours between both groups were insignificant. (p = 0.188, OR 0.6 [0.2, 1.3]). The median sodium correction rate within the first 24 hours was 0.27 mEq/L among patients with tests taken from the ED, whereas it was 0.1 mEq/L in the hospital ward group. (U= 3225.0, z = -4.08, p < 0.001, r = 0.27). The significantly higher sodium correction rate in the ED group potentially indicates earlier targeted intervention, although the small effect size (r=0.14) for LOS warrants further clinical interpretation and studies.

Keywords: *hyponatremia, urine chemistry, emergency department, inpatient care, sodium correction*

INTRODUCTION

Background/Rationale: Hyponatremia affects 15–30% of hospitalised patients and is associated with increased mortality, length of stay (LOS), and healthcare costs.[1,2] In hyponatremic patients, urine osmolarity and sodium concentration are ideally measured once fluid volume status and serum osmolarity are established. These tests reveal the specific causes of sodium loss, whether it is renal (with high urinary sodium) or extrarenal (low urinary sodium). High urine osmolarity reveals concentrated urine in patients with the syndrome of inappropriate antidiuretic hormone secretion. The European 2014 Clinical Practice Guideline on the diagnostic approach and treatment of hyponatraemia emphasises prompt urine chemistry to guide therapy.[3] However, in emergency conditions such as severe hyperglycaemia, hypovolemia, and symptomatic hyponatremia (i.e., seizures, coma and a reduced level of consciousness), immediate resuscitation and fluid correction take priority before urine tests are performed.[4] This leads to a delay in categorising hyponatremia, which is frequently initiated following ward admission. Delayed or incorrect differentiation in the aetiology of hyponatremia, such as between SIADH and hypovolemia, directly causes harm through inappropriate fluid management. This exacerbates hyponatremia, increases neurological and medical complications, prolongs recovery, and increases mortality risk.[3,4]

Objectives: This study described differences in the outcomes of performing urine osmolarity and sodium tests either early in the Emergency Department (ED) or later following ward admission. The outcomes measured were hospital LOS, 24-hour sodium correction rates, and modifications in the treatment

plan following the urine results. The location of the urine sample collected reflects the time at which the sample was collected. The median ED time at the location of this study was 6 hours.[5] It was hypothesised that ED urine osmolarity and sodium concentration clarify the category of hyponatremia early in its management, thus resulting in appropriate treatment, an improved sodium correction rate, and a shorter hospital LOS.

METHODOLOGY

Study design and setting

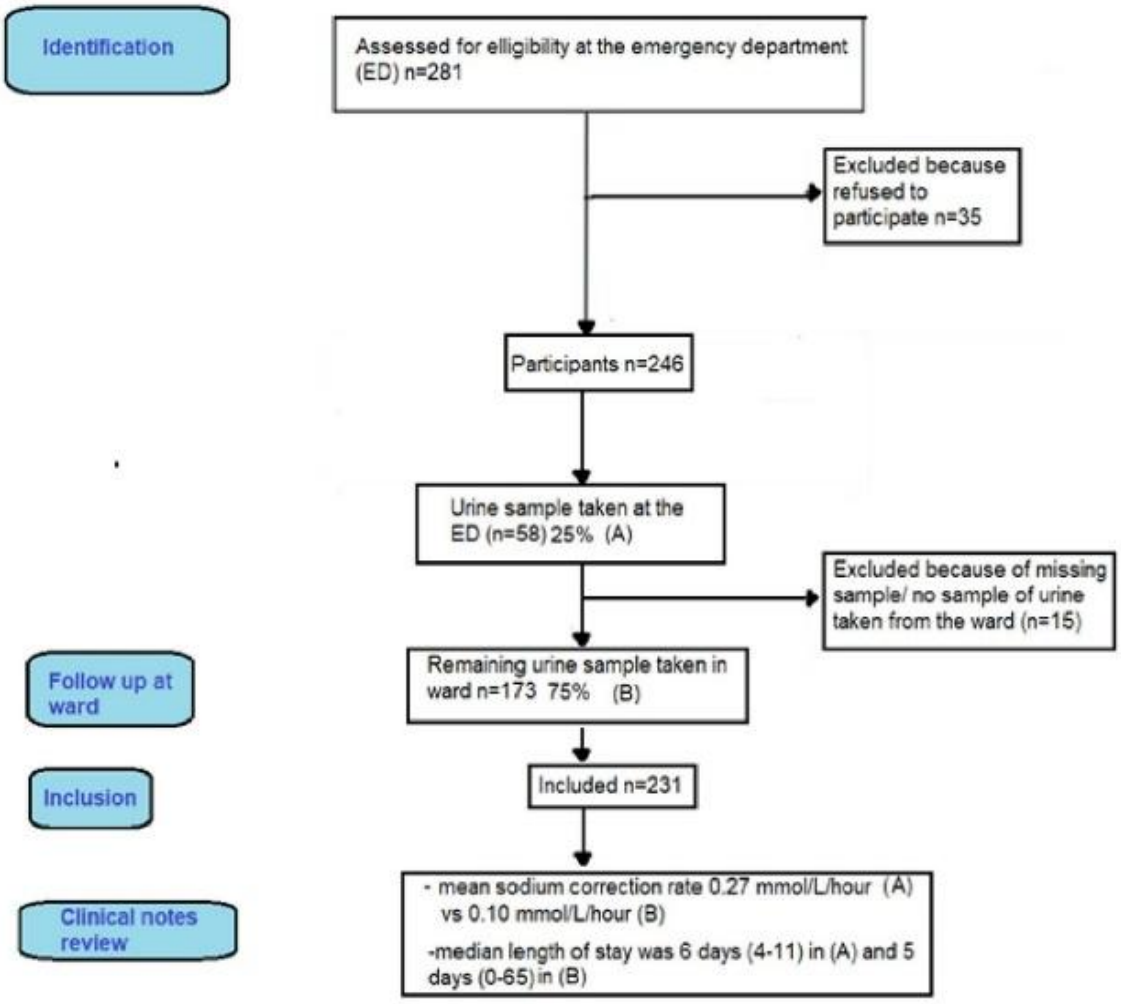
This prospective observational cohort study was conducted in a tertiary ED (Jan-Aug 2020). Each eligible patient was recruited and followed up within 24 hours of arrival at the ED.

Participants

Eligible patients were aged 18 years and above and admitted to the ward through the ED with recorded serum hyponatremia (< 136 mEq/L). Patients with no urine, i.e., end-stage renal failure or obstructive uropathy, and those discharged/deceased less than 24 hours after arrival were excluded. A single researcher initially screened all patients (>18 years, nontrauma etiology) at the critical or semicritical zones in the ED. Eligibility was identified among all patients planned for hospital admissions, where convenience sampling was employed. Convenience sampling was used during researcher shifts due to practical constraints, in which only a single researcher was employed.

The report of participants is included in the flow diagram below.

Figure 1: Results flow diagram



Patient involvement and consent

Consent was obtained from the patient or the patient's family if the patient was mentally incapacitated. Participation was voluntary, and personal data remained confidential.

VARIABLES

Exposures

As this was an observational study, the researcher did not influence any exposure, and the approach to hyponatremic patients followed the standard mode of therapy. Initially, the volume status of patients was determined via the clinical parameters of dehydration (sunken eyes, loss of skin turgidity, delayed capillary refill time, shock index, and reduced pulse volume) and ultrasonic measurement of inferior vena cava diameter. Patients were categorised into hypovolemic, euvolemic, and hypervolemic. Hypovolemic patients were resuscitated with normal saline, and hypervolemic patients were fluid restricted ± administered diuretics. Euvolemic patients were given either maintenance normal saline or no fluids intravenously. Blood samples were taken, and hyponatremic patients were selected after their serum sodium levels were measured. Saline (3%) was administered to severe hyponatremic patients with clinical manifestations of acute hyponatremia. (confusion, delirium, coma, seizure). Serum osmolarity was calculated on the basis of the blood results (2x serum sodium + glucose + urea mmEq/L), and patients were divided into hypertonic, isotonic, and hypotonic groups.

The attending physician was then instructed for urine osmolarity and sodium to be taken in the ED if the etiology of hyponatremia was unclear or if abnormal antidiuretic hormone (ADH) secretion was suspected. If the etiology of hyponatremia was clear and these diagnoses were not suspected at the ED, urine chemistry analysis was subsequently performed following hospital ward admission. (median ward admission time of 6 hours following arrival at the ED)[5]. The levels of ADH and urine osmolarity are not related to the severity of hyponatremia. However, it could guide the following management.[4] Following this decision, patients were further categorised into two groups: urine osmolarity and sodium levels taken in the ED or later in the hospital ward.

Outcomes

The first outcome documented was a management modification from the initial plan for the treatment of hyponatremia within 24 hours of arrival at the ED. Management modifications were objectively measured by documented treatment changes (e.g., fluid restriction to diuretics) within 24 hours of urine results. The two groups were compared to observe whether urine osmolarity and sodium concentration significantly altered the initial management. For example, in hypervolemic hyponatremia patients, fluid restriction was commenced and adjusted on the basis of urine chemistry results. Specifically, diuretics were used if urinary Na was less than 20 mEq/L, or fluid restriction alone was used if urinary Na was more than 20 mEq/L. This change would not have occurred if urine chemistry had not been taken in the ED. A patient data registry, discharge summary, and clinical records were obtained to note any management modifications. The second outcome recorded was the serum sodium correction rate within the first 24 hours following ED arrival (mEq/L/24 hours). This was accomplished by comparing the first recorded serum sodium level with the serum levels taken approximately 24 hours later. The therapeutic goal was to normalise sodium levels to 135–145 mEq/L with a correction rate not exceeding 8 mEq/L/24 hours to avoid complications such as osmotic demyelination syndrome (ODS) [3,6]. The third outcome recorded was total hospital LOS (time interval from ED registration to ward discharge).

Predictors

The volume status of the patient, either hypovolemic, euvolemic, or hypervolemic, as well as the age of the patient, can influence the outcome. The two main predictor variables observed were the time and location (ED vs ward) of urine collection for chemical analysis. Outcomes will depend on urine chemistry analysis and whether a new diagnosis for hyponatremia has been reached. This is predicted to influence the management of hyponatremia, hospital LOS, and 24-hour rate of sodium correction.

Potential confounders/effect modifiers

Potential confounders that can prolong the LOS are chronic causes of hyponatremia, such as respiratory, cardiovascular, renal, central nervous, musculoskeletal, malignancy, and drug-induced conditions. A confounder that can influence the sodium correction rate is the urgency with which it is corrected. This can occur when 3% saline is administered for symptomatic hyponatremia as per the protocol, accelerating the 24-hour sodium

correction rate.[7] The level of expertise and experience of the attending physician in ordering urine osmolarity and sodium levels influences the decision and categorisation of the two groups of hyponatremic patients studied.

Data sources/measurement

The physiological parameters, demographic characteristics, clinical symptoms, and outcomes of the patients were recorded via a standard data collection sheet. Once obtained, the serum sodium and urinary chemistry (osmolarity and sodium levels) data were sent to the biochemistry laboratory in the hospital. The results and locations of these tests were retrieved from the hospital's OMRs. Although urine chemistry data were obtained in the ED, the same patient's urine chemistry data in the ward were not analysed. The hospital LOS, including the time and date of admission and discharge and alterations in management, was recorded from the data registry, discharge summary, and clinical records.

Bias

A single investigator was employed to collect the data. While convenience sampling was used, an attempt to mitigate bias was made by transparent reporting and sample collection, as well as ensuring coverage across shifts (weekday/weekend, day/night) and diverse patient demographics. Off-hour patients were identified through medical records the following day.

Study size

Sample size determination was performed via a standard two-proportion power calculation based on the following formula:

$$n=(Z\alpha/2+Z\beta)2\times[p1(1-p1)+p2(1-p2)](p1-p2)2n=(p1-p2)2(Z\alpha/2+Z\beta)2\times[p1(1-p1)+p2(1-p2)]$$

where:

- $Z\alpha/2Z\alpha/2$ = critical value for a two-sided test at significance level α ($Z = 1.96$ for $\alpha = 0.05$)
- $Z\beta Z\beta$ = critical value for power ($1 - \beta$) ($Z = 1.282$ for 90% power)
- $p1p1$ and $p2p2$ = expected proportions in the comparison groups

The calculation was parameterised via published data on hyponatremia correction rates, where the prevalence of undercorrection (<6 mEq/L/24 h) was 38% ($p_1 = 0.38$) versus normal/overcorrection (≥6 mEq/L/24 h) at 62% ($p_2 = 0.62$).[8] This comparison served as the primary outcome for hospital length of stay (LOS) analysis. With $\alpha = 0.01$ (99% confidence level) and $\beta = 0.10$ (90% power), the minimum required sample size was 122 participants per group.

Quantitative variables

The quantitative variables identified were demographic data such as age, sex, and ethnicity. Data were collected from the patients' records and OMRs. Simple descriptive analysis, i.e., mean and standard deviation, was employed.

Statistical methods

Normality was assessed via Shapiro–Wilk tests. Nonnormally distributed data (LOSs, sodium correction rates) were analysed with Mann–Whitney U tests. A chi-square test was used to calculate categorical variables for differences in patient outcomes between the two groups with a normal distribution. Multivariable linear regression adjusted for confounders (age, severity, volume status, 3% saline use). The IBM Statistical Package for the Social Sciences (SPSS version 22.0) was utilised to analyse demographic data and clinical characteristics. Missing or incomplete data were excluded from the analysis.

RESULTS

Table 1: Demographic data and clinical characteristics (n=231)

Mean age, SD	60.6 ± 19.4 years old
Gender, n (%)	
Male: Female ratio	142:89 (61.5%:38.5%)
Race, n (%)	
Malay	112 (48.5%)
Chinese	95 (41.1%)
Indian	18 (7.8%)
Others	6 (2.6%)
Mean serum sodium on admission, mEq/L± SD	128.4 ± 6.5

Volume status	
Hypovolemic	112
Euvolemic	97
Hypervolemic	22
Type of hyponatremia, n (%)	
Hypotonic	100 (43.4%)
Isotonic	84 (36.4%)
Hypertonic	47 (20.3%)
Aetiology of hyponatremia, n (%)	
Gastrointestinal	70 (30.3%)
Respiratory	46 (19.9%)
Cardiovascular	30 (13%)
Renal	19 (8.2%)
Others/multifactorial	66 (28.6%)
Correction rate (over 24 hours)	
Under corrected (< 0.3 mEq/L/hour)	160 (69.3%)
Over corrected (>0.5 mEq/L/hour)	30 (13%)
Correction as recommended (0.3-0.5 mEq/L/hour)	41 (17.7%)
Initial management	
Normal saline	172 (74.4%)
Other crystalloids ± dextrose	14 (6.1%)
Fluid restriction ± Diuretics	38 (16.5%)
3% Hypertonic saline	7 (3%)

Table 2: Comparison between two groups of urine samples sent from the ED and those sent from the ward

Target studied	**Urine samples sent from ED, n=58**	**Urine samples sent from the ward, n=173**	**P value**
Severity	**n (%)**	**n (%)**	**0.98**
Mild (130-135 mEq/L),	10 (17%)	123 (71%)	
Moderate (125-129 mEq/L)	10 (17%)	37 (21%)	
Severe (<125 mEq/L)	38 (66%)	13 (8%)	
Tonicity	**n (%)**	**n (%)**	
Hypotonic	43 (74%)	57 (33%)	
Isotonic	8 (14%)	76 (44%)	
Hypertonic	7(12%)	40 (23%)	
Initial management	**n (%)**	**n (%)**	**0.188**
NS/balanced solution	44	142	
3% NS	7	0	
Fluid restriction± diuretic	7	31	
24-hour correction rate	**mEq/L/hour**	**mEq/L/hour**	**<0.001**
Median	0.27	0.1	
IQR	0.08 - 0.51	0.02 – 0.22	
Range	0-1.45	0-3.2	
Hospital length of stay	**days**	**days**	**0.038**
Median (IQR)	6 (4 – 11)	5 (3 – 8.5)	
Range	1 – 32	0 – 65	

NS: normal saline, FR±diuretic: fluid restriction ± diuretics (frusemide)

Descriptive Data

The cohort (n=231) had a mean age of 60.6±19.4 years and an average sodium concentration of 128.4±6.5 mEq/L. Only 58 (25.1%) of the 231 patients with hyponatremia were tested for urine sodium and osmolality in the ED (Table 1). Management modifications were objectively measured by documented treatment changes (e.g., fluid restriction to diuretics) within 24 h of urine results. Thirty-five patients refused participation, 15 had missing data, or no urine samples were taken in the ward (Figure 1). The demographic and clinical data are presented in Table 1.

All patients with hyponatremia (hypotonic, isotonic, or hypertonic) received normal saline 0.9% or fluid restriction ± diuretics as initial management,

depending on their clinical signs. Undervolume, dehydration, or normovolemic hyponatremia was managed with normal saline at 0.9%. Hyponatremia patients with fluid overload were treated with fluid restriction ± diuretics, regardless of the type of hyponatremia. Symptomatic hyponatremia patients (seizure, coma) received 3% saline, and all of the patients had hypotonic hyponatremia, in which urine samples were taken in the ED.

Outcome data

The median sodium correction rate was 0.13 mEq/L/hour (0.04 - 0.30). Fifty-eight percent of patients had mild hyponatremia (130–134 mEq/L), and the median hospital length of stay (LOS) was 5 days.

Main results

There was a significant difference in the hospital LOS between the group with urine samples sent from the ED and those with urine samples sent from the ward. (p = 0.038), median LOS 6 days (4-11), and 5 days (0-65). Outliers of range in LOS (up to 65 days) reflecting comorbidities of the patient and not solely dependent on the correction of hyponatremia.

This test also revealed a significant difference in the sodium correction rate between the two groups (p <0.001). Sodium correction: 0.27 mEq/L/hour (tests taken in the ED) vs. 0.10 mEq/L/hour (tests taken in the ward)

There was no significant association in management change between sending urine samples from the ED and the ward. (p = 0.188). A similar nonsignificant association was obtained in the subgroup analysis on management modifications among patients with moderate and severe hyponatremia. (p = 0.94)

DISCUSSION

The low prevalence of hyponatremic patients who underwent early urine chemistry testing has also been reported in other studies, ranging from 19% to 31%. This is partially due to the management of hypovolemia, dehydration, and symptomatic hyponatremia (seizures, loss of consciousness), which takes precedence over urine chemistry analysis in the ED. [5]

Sodium correction rates

Earlier ED urine testing was associated with significantly higher sodium correction rates (0.27 vs. 0.10 mEq/L/hour), approximating rates (0.3–0.5 mEq/L/hour).[3] This may reflect earlier targeted interventions. This was associated with a shorter LOS and a better survival rate.[10] Undercorrection of hyponatremia was associated with a significant increase in mortality and LOS. [11] However, the bias factor arises when a large number of patients with mild hyponatremia, n= 123 (71.1%), among samples taken from the ward, compared with only 10 (17.2%) taken at the ED, significantly affected the average rate of sodium correction. Limited evidence is available on the associations between the use of urine sodium and osmolality tests and the sodium correction rate. When subgroups of patients with hyponatremia, i.e., mild, moderate, and severe, were compared, the difference in management modifications between urine samples taken from the ward and those taken from the ED was not significant (p=0.45). This was due to the removal of confounding factors, such as severe hyponatremia, which also affects management modifications and hospital LOS. Early detection of the cause of hyponatremia can help clinicians manage the disease more accurately. However, urgent correction in cases of dehydration and symptomatic hyponatremia does not require urine chemistry analysis.

A multivariable linear regression analysis, adjusting for age, severity of hyponatremia, volume status, and comorbidities, revealed that performing urine osmolarity/sodium tests in the ED was associated with a 0.17 mEq/L/hour higher sodium correction rate (95% CI: 0.10–0.24; p< 0.001) than tests performed in the ward. Severe hyponatremia and hypovolemic status were also independently associated with faster correction rates

Hospital length of stay

The overall difference in the median LOS between the two groups was significant (p=0.038). The increase in hospital LOS among patients whose urine samples were sent from the ED was due to a higher percentage of severe hyponatremia, 38 (65.5%) vs 13 (7.5%), in which attending physicians were more inclined to perform urgent urine chemistry in the ED (Table 2). The hospital LOS was longer than that reported in previous studies. However, it is related to a slow correction rate and the presence of hyponatremia.[2,12] No previous study has compared the location of urine chemistry with the hospital LOS. However, this finding indirectly implies that misdiagnosis due to delayed urine chemistry results in a longer hospital LOS. Confounding factors such as underlying comorbidities (diabetes, cancer, and renal failure) and disease complexity were more profound in patients with severe hyponatremia, which influences hospital LOS. A

lack of medical beds, backlog, and ED overcrowding at the location of the study also influenced the LOS.[5,13]

While the LOS was significantly shorter in the ward group (5 vs. 6 days), the 1-day difference was of uncertain clinical significance given the small effect size (r=0.14). This likely reflects the higher acuity of the ED group (66% had severe hyponatremia vs. 7.5% in the ward group). Furthermore, when subgroups of patients with hyponatremia, i.e., mild and moderate hyponatremia (125--135 mEq/L), were compared, there was no significant difference in hospital LOS between the two groups. (p=0.44)

Change in management

Urine chemistry analysis in the ED revealed no significant effect on the initial management of hyponatremia (p=0.188). This primarily stems from the fact that acute therapeutic interventions are dictated by the patient's clinical severity and hemodynamic assessment rather than the specific etiology suggested by urine chemistry. For example, in hypovolemic hyponatremia, urgent isotonic saline resuscitation was initiated irrespective of the urinary sodium or osmolality results. Fluid restriction was implemented upon diagnosis of hypervolemic hyponatremia. Intravenous diuretics (furosemide) were administered for complications such as pulmonary edema or congestive heart failure. Severe neurological manifestations (e.g., seizures, coma) in acute hyponatremia warrant urgent hypertonic saline (3%) administration, overriding any urine chemistry findings.

Management initiated in the ED typically continued upon ward admission, with subsequent adjustments guided primarily by evolving clinical signs and serial serum sodium measurements. The absence of standardised institutional protocols further contributed to the frequent underutilisation of urine chemistry results in guiding therapeutic decisions during the acute phase.

Nevertheless, urine chemistry plays a more definitive role in diagnosing euvolemic hyponatremia, particularly in patients lacking severe symptoms where immediate treatment is withheld. In these cases, findings of high urine osmolality and sodium strongly suggested SIADH. However, even with this diagnosis, the subsequent management (fluid restriction) aligns with the standard approach for hypervolemic states on the basis of volume status. Therefore, the principal utility of ED-obtained urine samples appears to lie in guiding the ongoing management of stable, euvolemic hyponatraemic patients after they transition to the ward setting.

Clinical implications

Patients in the cohort without ED-obtained urine chemistry analysis predominantly presented with mild hyponatremia (serum sodium 130–135 mEq/L). Initial management for this group typically addresses both hyponatremia and concomitant hypovolemia concurrently. Establishing the precise etiology of hyponatremia has proven challenging in the context of compromised volume status, limiting diagnostic clarity.[14] Existing evidence supports the utility of prompt urine chemistry evaluation in guiding appropriate hyponatremia management.[15] Furthermore, in the absence of severe or moderately severe symptoms, urine chemistry analysis is valuable for informing targeted therapeutic decisions within the ED setting.[16] While higher correction rates suggest potential benefits from ED testing for specific patient subgroups, resource allocation implications necessitate careful consideration, given its limited association with a reduced length of stay.

Limitations

The timing of urine chemistry collection is influenced by clinical priorities, particularly the need to stabilise patients and correct dehydration, which can delay sample collection and introduce variability. Consequently, a disproportionate number of urine chemistry tests were obtained from ward settings, potentially leading to selection bias. While off-hour data collection was employed to mitigate this bias, residual confounding remains. Additionally, confounding by indications was present, as urine testing in the emergency department (ED) was more likely to be performed in patients with severe hyponatremia. Multivariable adjustments were made for 3% saline administration and comorbidities when analysing sodium correction rates but not for length of stay (LOS). Most urine sodium tests obtained in the ED were from patients with severe hyponatremia (serum sodium <125 mEq/L), indicating the need for aggressive therapy.

In contrast, patients with milder cases (n=36, 65.5%) had urine tests primarily performed after admission to the ward (n=13, 7.5%) (Table 2). This distribution likely skews the correction rate and LOS data, making outcomes more pronounced in severe cases. Furthermore, the presence of comorbidities and concurrent medications may affect urine chemistry results; however, this confounding factor cannot be

fully controlled, given the high prevalence of underlying conditions in the study population.

CONCLUSION

Urine testing in the ED was associated with significantly higher sodium correction rates. However, hospital LOS reduction was clinically insignificant, given that only a one-day reduction and multiple confounding factors (i.e., comorbidities) influenced the results, which is unlikely to affect clinical practice. Nevertheless, it may reflect diagnostic efficiency. Early testing may optimise sodium correction in severe hyponatraemia patients, although routine implementation requires cost–benefit analysis. Future studies should identify specific patient subgroups (either hypovolemic, euvolemic or hypervolemic hyponatremia) that benefit most from ED urine chemical testing.

COMPLIANCE WITH MANUSCRIPT WRITING GUIDELINES

This article complies with the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) Statement for cohort studies.

CONFLICT OF INTEREST

The author(s) declare that there are no conflicts of interest related to the study, authorship, and/or publication of this article.

ETHICAL APPROVAL

Ethical approval was obtained from the Research Ethical Committee, The National University of Malaysia, with the code project FF-2020-051. from 1/02/2020 to 01/08/2020

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CORRESPONDENCE

Dr. Nik Azlan Nik Muhamad
MMed (Emergency Medicine)
Department of Emergency Medicine,
Universiti Kebangsaan Malaysia Medical Centre (UKMMC)
Jalan Yaacob Latif, Bandar Tun Razak,
56000 Kuala Lumpur, Wilayah Persekutuan,
Malaysia.
ORCID iDs: 0000-0003-4673-7385
Email: nikazlanmuhamad@hotmail.com

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