ORIGINAL ARTICLE

Sonographic Optic Nerve Sheath Diameter as a Guide for Correction of Hyponatremia in the Emergency Department: A Cross-sectional Study

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Abstract

Background: Monitoring sodium levels during the correction of hyponatremia is essential. There is cell swelling due to the movement of water from extracellular to intracellular by osmotic effect in hyponatremia. The cellular swelling in a closed space causes increased intracranial pressure (ICP). The raised ICP correlates with the optic nerve sheath diameter (ONSD). So, the research question was whether the ONSD can be used as a guide for the correction of hyponatremia.

Methods: It was a prospective observational study conducted on patients with serum sodium below 135 mEq/L presented to the emergency department (ED). The ONSD was measured at the time of presentation and discharge of the patient. The receiver operating characteristic curve (ROC) and area under the curve (AUC) were used to test the predictive ability of the ONSD to diagnose hyponatremia.

Results: A total of 54 subjects were included in the study. The mean sodium level was 109.3 mEq/L at presentation. The mean ONSD on the right side was 6.24 ± 0.71 mm and on the left side was 6.26 ± 0.64 mm at presentation to ED. The mean ONSD on the right side was 5.81 ± 0.58 mm and on the left side was 5.79 ± 0.56 mm at discharge. The ONSD was not able to predict the sodium level measured both by laboratory and POC methods.

Conclusion: The ONSD failed to predict the sodium level in patients with hyponatremia during the correction. The change in ONSD did not correlate with the change in sodium level.

Keywords: Electrolyte imbalance, Emergency department, Hyponatremia, Optic nerve sheath diameter, Ultrasonography.

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Highlights

There is increased intracranial pressure (ICP) in hyponatremia due to the osmotic effect. There is a correlation between optic nerve sheath diameter (ONSD) with ICP. The change in ONSD did not correlate with the change in sodium level during the correction of hyponatremia.

Introduction

The prevalence of hyponatremia in the emergency department (ED) is about 3%.[1] Monitoring serum sodium levels during the correction of hyponatremia is essential to prevent the development of central pontine myelinolysis or osmotic demyelination syndrome.[2,3] As the laboratory testing for electrolytes takes longer, the blood gas analysis machine is used as a point of care (POC) for this to monitor the correction of hyponatremia in ED. Moreover, repeated pricking of the patient for sample collection and the high cost of the blood gas analysis are the disadvantages.

In hyponatremia, water movement from extracellular to intracellular space due to an osmotic gradient leads to cell swelling. The symptoms are primarily neurological because there is less space for the swollen cells in the cranial cavity leading to increased ICP.[4] There is continuity of the meninges and subarachnoid space around the optic nerve into the orbit.[5] The raised ICP can be monitored indirectly by measuring the ONSD using ultrasonography.[6] There is a correlation between ONSD with a change in ICP.[7–13] The ONSD is a reliable predictor of elevated ICP.[14] The ONSD has been used for the neurological monitoring of ICU patients.[9,10,15] Sonographic ONSD measurement can be used for ICP monitoring when invasive methods are not available or contraindicated.[16] The ONSD measured in computed tomography (CT) of the head is a surrogate for ICP in moderate to severe traumatic brain injury.[17] When compared to CT, ocular sonography demonstrates better diagnostic test accuracy for the detection of increased ICP.[18]

There is a change in ONSD in patients who presented with hyponatremia.[4] To our knowledge, there is no literature on changes in ONSD during the correction of hyponatremia. So, we wanted to study whether ONSD measurement can be used for discharging a patient with hyponatremia from the ED. The

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objective of the study was to assess ONSD as a guide for correcting hyponatremia in ED.

Methods

We conducted a prospective observational study in the ED of a tertiary care teaching hospital after approval from the Institutional Ethics Committee (IEC/AIIMS BBSR/PG Thesis/2019-52) and registered prospectively at the Clinical Trials Registry – India (CTRI/2020/05/025306). We obtained consent from the authorized person of the patient. We included patients over 18 years of age with serum sodium below 135 mEq/L and clinical features of acute hyponatremia presenting to the ED. We excluded the patients with known or suspected raised ICP (head injury, intracranial space-occupying lesion, and stroke), known psychiatric illness, hepatic/uremic encephalopathy, hemodynamically unstable, hypervolemic hyponatremia, and ocular injury/pathology from the study.

We calculated the sample size by taking the mean value of ONSD as 0.544 cm in pretreatment with a standard deviation (SD) between 0.06 and 0.507 cm in posttreatment with a standard deviation of 0.07 with absolute precision of 5% and power of 80%, the sample size (*N*) was 28.[4]

We diagnosed hyponatremia with POC testing of electrolytes by blood gas machine (Siemens Rapidpoint, Siemens Health Care Diagnostics Inc., NY, USA) and clinical features. We recorded the heart rate, blood pressure, and oxygen saturation. We measured the ONSD of both eyes and assessed the level of sensorium. We measured the ONSD using a high-frequency linear ultrasonography probe (6–13 MHz, Sonosite M-turbo, Fujifilm, Inc., Japan) with eyes closed in a 25–30° head-up position. We kept the machine in the ophthalmic setting to reduce the ultrasound power and sonic output. By using hypoechoic lines as a reference, the ONSD measurement was carried out at 3 mm posterior to the optic disc on the transverse plane.[19] We collected the data for this study after practicing at least 80 individual measurements of ONSD to reduce the error.[20] We measured the level of sensorium using the Richmond agitation sedation score (RASS) and Glasgow coma scale score (GCS).[21,22] Then we started correcting hyponatremia as per the POC serum sodium level. We sent blood samples to the central laboratory for complete blood count, kidney function test, and liver function tests. We monitored the patients for vitals, serum sodium level, GCS, and RASS. We discharged the patients as per the clinical improvement and correction of hyponatremia by POC testing of sodium level. We measured the ONSD at the time of discharge.

All the data were collected and entered in a Microsoft Excel sheet and further analyzed in Statistical Package for the Social Sciences (SPSS), version 27 (IBM Corporation, New York, United States). Categorical data were expressed in numbers and percentages while continuous variables were expressed in mean and standard deviation. The normality of the continuous variables was checked using the Shapiro–Wilks test and depending on the normality statistical tests were applied. Comparison between two means between the same groups was assessed using paired sample *t*-test or related sample Wilcoxon signed rank test. Correlation between two continuous variables was assessed using the Pearson correlation test using a scatter plot and correlation coefficient value. The predictive ability of ONSD to correctly diagnose moderate to severe hyponatremia was assessed using the receiver operating characteristic curve (ROC) and represented using the area under the curve (AUC). A *p*-value less than 0.05 was considered statistically significant.

Table 1: Baseline characteristics of the study population (*N* = 54)

Variables	*Mean/frequency*	*SD/percentages*
Age (mean and SD)	58.65	13.11
Gender (frequency and %)		
Male	29	53.7
Female	25	46.3
Moderate hyponatremia*	4	7.4
Severe hyponatremia**	50	92.6

*Sodium level: 120–129 mEq/L; **Sodium level: <120 mEq/L

Table 2: Presenting complaint and comorbidities among the study participants (*N* = 54)

Symptoms	*N (%)*
Altered sensorium	30 (55.6)
Vomiting	24 (44.4)
Seizure	8 (14.8)
Weakness	5 (9.3)
Hiccups	3 (5.6)
Headache	2 (3.7)
Nausea	2 (3.7)
Reeling	2 (3.7)
Shortness of breath	2 (3.7)
Others*	5 (9.3)

*Includes one case each of vertigo, slurred speech, anasarca, abdominal pain, and diarrhea

Results

A total of 54 subjects were included in the study. Twenty-nine patients were male. The mean age of the study population was 58.6 years (SD ± 13.1) (Table 1). The most common presenting symptoms were altered sensorium, vomiting, and seizure (Table 2). Most of the patients were having severe hyponatremia (Table 1). The vital parameters, GCS and RASS are presented in Table 3. The mean sodium level was 109.3 mEq/L at presentation (Table 4). The mean random blood sugar (RBS) was 129.7 mg/dL (SD ± 41.01) and blood urea was 56.60 mg/dL (SD ± 61.83) at presentation. The RBS and urea levels at presentation and after the correction of hyponatremia were comparable (Table 4).

The mean ONSD on the right side was 6.24 ± 0.71 mm and on the left side was 6.26 ± 0.64 mm at presentation to ED (Table 4). After the treatment of hyponatremia, the mean ONSD on the right side was 5.81 ± 0.58 mm [95% confidence interval (CI): 0.27–0.59, $p < 0.001$], and on the left side was 5.79 ± 0.56 mm (95% CI: 0.29–0.64, $p < 0.001$). There was a statistically significant difference in ONSD before and after the treatment of hyponatremia. The mean RASS at the presentation was –1.41 (SD ± 1.53) and after treatment was –0.52 (SD ± 0.74). The mean GCS was 11.88 (SD ± 2.09) at presentation and 13.83 (SD ± 1.46) after treatment of hyponatremia. There was a statistically significant improvement in both RASS (95% CI: From –1.25 to –0.51, $p < 0.001$) and GCS (95% CI: From –2.5 to –1.3, $p < 0.001$) after treatment (Table 3).

As per the ROC, the ONSD was not able to predict the moderate to severe hyponatremia measured both by laboratory and POC methods (Table 5 and Figs 1 and 2). The AUC for the right ONSD was 0.632 [95% CI: 0.414–0.85, standard error (SE) = 0.11, $p = 0.28$] and the left ONSD was 0.733 (95% CI: 0.533–0.932, SE = 0.10, $p = 0.058$)

Table 3: Comparison of clinical parameters before and after the treatment of hyponatremia

Parameters	*Before treatment (Mean ± SD)*	*After treatment (Mean ± SD)*	*Mean difference with 95% CI*	*p-value*
SBP	129.98 ± 24.61	127.67 ± 23.56	2.30 (–4.9 to 9.6)	0.528
DBP	78.77 ± 17.13	76.51 ± 13.13	2.25 (–2.0 to 5.5)	0.292
HR	86.23 ± 15.47	82.47 ± 18.51	3.76 (–2.4 to 9.9)	0.226
SpO_2	97.70 ± 3.44	98.14 ± 1.82	–0.44 (–1.6 to 0.7)	0.693*
GCS	11.88 ± 2.09	13.83 ± 1.46	–1.95 (–2.5 to –1.3)	<0.001*
RASS	–1.41 ± 1.53	–0.52 ± 0.74	–0.89 (–1.25 to –0.51)	<0.001

*Related sample Wilcoxon signed rank test was used to calculate *p*-value and for rest of the variables, paired sample *t*-test was used. DBP, diastolic blood pressure; GCS, Glasgow coma scale score; HR, heart rate; RASS, Richmond agitation sedation score; SBP, systolic blood pressure; SpO_2, peripheral oxygen saturation

Table 4: Comparison of ONSD and sodium levels before and after the treatment of hyponatremia

Parameters	*Before treatment (Mean ± SD)*	*After treatment (Mean ± SD)*	*Mean difference with 95% CI*	*p-value*
ONSD (right)	6.24 ± 0.71	5.81 ± 0.58	0.43 (0.27–0.59)	<0.001
ONSD (left)	6.26 ± 0.64	5.79 ± 0.56	0.46 (0.29–0.64)	<0.001
Na^+ by POC	109.30 ± 10.21	121.52 ± 6.59	–12.21 (–15.3 to –9.1)	<0.001
Na^+ by laboratory	112.79 ± 7.38	121.67 ± 6.94	–8.87 (–11.6 to –6.12)	<0.001
Urea*	56.60 ± 61.83	63.80 ± 84.29	–7.20 (–22.3 to 7.9)	0.629
RBS*	129.73 ± 41.01	131.97 ± 53.51	–2.24 (–18.2 to 13.7)	0.774

*Related sample Wilcoxon Signed rank test was used to calculate *p*-value and for rest of the variables, paired sample *t*-test was used. ONSD, optic nerve sheath diameter; POC, point of care; RBS, random blood sugar

Table 5: Predictive ability of ONSD for hyponatremia

	AUC	*95% CI*	*SE*	*p-value*
Laboratory method				
ONSD (right)	0.632	0.414–0.850	0.11	0.282
ONSD (left)	0.733	0.533–0.932	0.10	0.058
POC method				
ONSD (right)	0.519	0.357–0.681	0.083	0.817
ONSD (left)	0.538	0.380–0.696	0.080	0.643

ONSD, optic nerve sheath diameter; POC, point of care

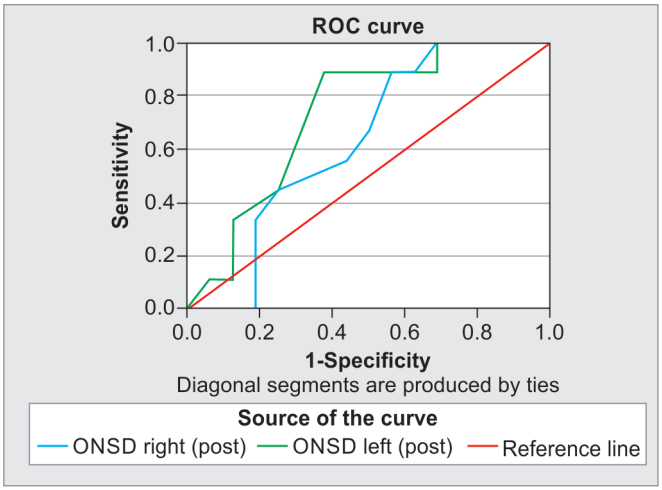

Fig. 1: ROC curve analysis of the predictive ability of ONSD for moderate to severe hyponatremia measured by the laboratory method

for the sodium level measured by the laboratory method. The AUC for the right ONSD was 0.519 (95% CI: 0.357–0.681, SE = 0.083, $p = 0.817$) and the left ONSD was 0.538 (95% CI: 0.380–0.696, SE = 0.08, $p = 0.643$). The correlation coefficient was 0.097 and 0.014 for right and left ONSD, respectively (Table 6). The scatter plot shows no correlation between the change in sodium level and ONSD (Fig. 3). The median length of hospital stay was 26.56 ± 9.57 hours with a range of 9–48 hours. None of the patients had revisited the ED due to deterioration of the sensorium or hyponatremia within one week after discharge.

Discussion

The ONSD failed to predict the sodium level measured by both laboratory and POC methods. The change in sodium level was not correlated with ONSD. The mean age of the patients was 58 years. This is similar to a study in India by Padhi et al. where the mean age of the participants admitted to the intensive care unit was 60 years.[23] But the mean age of the patients was 74 years in a study conducted in Sweden.[1] Most of the patients were having altered sensorium and vomiting in our study, but a study by Babaliche et al. found vomiting, confusion, and seizures were the most common presenting complaints.[24] Neurological symptoms such as dysarthria, motor function deficits, sensory loss, convulsions, vertigo, balance disorder, and headache followed by fatigue and abdominal pain were the main presenting symptoms in a study conducted by Olsson et al.[1]

In a study by Demir et al., the ONSD was greater in patients with hyponatremia. In the pretreatment period, the mean ONSD of symptomatic hyponatremia patients was significantly greater than

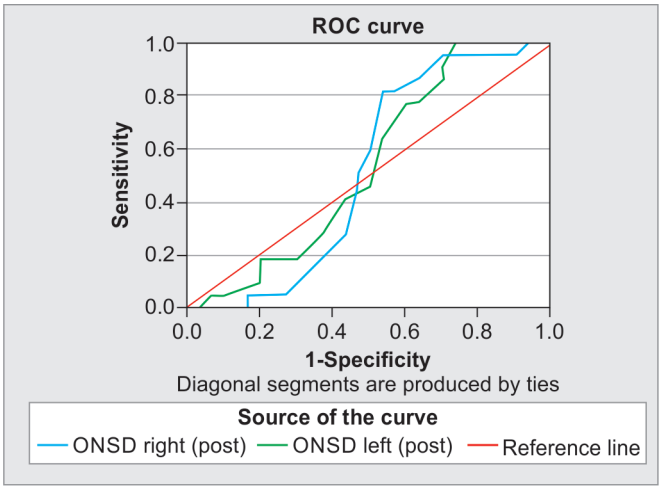

Fig. 2: ROC curve analysis of the predictive ability of ONSD for moderate to severe hyponatremia measured by point of care method

that of asymptomatic patients. The ONSD decreased significantly with the treatment of hyponatremia. They had not assessed symptomatic improvement during the treatment and not measured the sodium levels at the completion of treatment.[4]

There was a statistically significant change in the ONSD at presentation to after-treatment of sodium level in our study. The RASS and GCS showed a statistically significant change from before treatment to after treatment of hyponatremia. The blood sugar and urea were within the normal range in our study. So, it was not affecting the sodium level and the correction factor was not required.[25] Non-optimal correction especially under-correction (<6 mEq/L at 24th hour) of sodium has increased mortality in patients with severe hyponatremia.[26] We have corrected the sodium level optimally. There is a strong correlation between sodium levels measured by the POC and laboratory methods.[27] We have discharged the patients based on sodium levels measured by POC and clinical improvement.

The median hospital length of stay was 26.5 hours in our study. But in a study by Olsson et al., it was 7 days (IQR: 4.0–10.8) for the patients having sodium levels <120 mmol/L.[1] The median length of stay was 5 days in a retrospective study of profound hyponatremia patients.[28]

Strength and Limitation

To the best of our knowledge, this was the first study on the change of ONSD in relation to the correction of hyponatremia. This was a single-center study. The patients were not differentiated as acute or chronic hyponatremia.

Conclusion

The ONSD failed to predict the sodium level in patients with hyponatremia during the correction. The change in ONSD did not correlate with the change in sodium level. So, the ONSD cannot be used as a guide for correcting hyponatremia.

Orcid

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Table 6: Correlation of ONSD with change in sodium level

Correlation of change in sodium level with	*Correlation coefficient*	*p-value*
ONSD (right)	–0.097	0.653
ONSD (left)	0.014	0.948

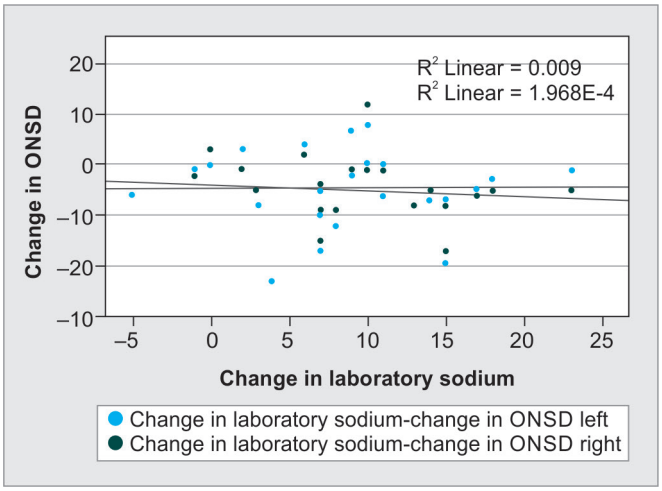

Fig. 3: Correlation between change in ONSD and sodium level before and after treatment

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