EDITORIAL

Optic Nerve Sheath Diameter and Sodium Levels: A Friend of a Friend is Still a Stranger

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The article titled "Sonographic optic nerve sheath diameter (ONSD) as a guide for correction of hyponatremia in the emergency department (ED): A cross-sectional study" investigated whether the ONSD can be used as a guide for the correction of hyponatremia in the ED.[1] The authors conducted a prospective observational study on 54 patients with serum sodium levels below 135 mEq/L who presented to the ED. They measured the ONSD at presentation and discharge and assessed its ability to predict changing sodium levels during the correction of hyponatremia using the receiver operating characteristic curve and area under the curve. They also checked the correlation between serum sodium levels and ONSD. The authors found that there was a change in ONSD in patients with hyponatremia at presentation, but the ONSD failed to predict the sodium level in patients with hyponatremia during the correction. Moreover, the change in ONSD did not correlate with the change in sodium level. Therefore, the authors concluded that ONSD measurement cannot be used as a guide for discharging a patient with hyponatremia from the ED.

The article highlights the importance of monitoring serum sodium levels during the correction of hyponatremia to prevent neurological complications such as central pontine myelinolysis or osmotic demyelination syndrome (ODS). As water moves from extracellular to intracellular spaces in hyponatremia, cell swelling occurs, leading to increased intracranial pressure (ICP).[2] Optic nerve sheath diameter has been extensively used for the estimation of ICP.[3–5] The present study is the first study to evaluate the relationship between serum sodium and ONSD. The authors hypothesized that with the correction of hyponatremia, the swelling of neurons decreases which may lead to a decrease in ICP which will be reflected by a decrease in optic nerve sheath diameter. After hyponatremia has been corrected, this may then be used to direct safe ED discharges. In principle, the theory examines a significant knowledge gap and has strong biological plausibility. If true, it could have improved patient safety during ED discharge.

The sheath of the optic nerve is made up of the Dura, Arachnoid, and Pia mater, which border a small amount of cerebrospinal fluid (CSF) in the subarachnoid space. Because the optic nerve is surrounded by CSF, which is connected to the brain's ventricular system, an increased ICP is thought to cause transmission of force through these spaces, leading to distention of the ONSD.[5] Optic nerve sheath diameter measurements using ultrasonogram (USG) are simple, easy to learn, non-invasive, point-of-care ocular ultrasound, and have multiple applications for the detection of raised ICP in common disorders like stroke, neuro infection, and neurotrauma and also in acute mountain sickness, posterior reversible encephalopathy syndrome, and post-cardiac arrest patients.[6–8] Compared to computed tomography (CT) it has a sensitivity of 95.6% and a specificity of 92.3%.[9] It can be performed using high-frequency linear probe (frequency of 7.5 MHz or higher) which is commonly available as part of portable ultrasound. Thus, it appears as an attractive tool for bedside monitoring of raised ICP. However, it has its limitations. Reported cut-off values for diagnosis of elevated ICP are inconsistent.[8] It may be due to the different patient populations, the ONSD range of normal in the general populace, USG machine settings, transducer placement (variation in the true axial placement of the probe), and ONSD delineation.[9] The ONSD measurement is operator dependent. Variability in USG marker positions used to delineate the optic nerve sheath results in different ONSD values.[8] Minor changes in ONSD values lead to different sensitivities to changes in ICP making it less reliable.[8] Stevens et suggested placement of ultrasound markers at the transitions from the single dark region to the hyperechoic retrobulbar fat or the outer edges of the hyperechoic-striped bands as the measurements with these areas provided the highest sensitivity.[8] Estimating interobserver and intraobserver agreements for the current research could have reduced observer bias, which could have had an impact on the study's findings. The authors have however used experienced operators and well-defined reference lines for ONSD measurements. The sample size was calculated based on a 0.04 cm change in ONSD (0.544 cm in pretreatment with a standard deviation of 0.06 and 0.507 cm in posttreatment with a standard deviation of 0.07) which makes precision measurement a necessity.

The study included a predominantly older population (mean age 54 years) reflecting the usual epidemiology of hyponatremia and the majority has profound hyponatremia (50 patients). The

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study could have been improved by data on the presence of comorbidities like alcohol use, liver failure, malnutrition, and AIDS which are risk factors for ODS. The patients' sensorium was monitored with Glasgow Coma Scale (GCS) and Richmond Agitation-Sedation Scale (RASS). The mean sodium pretreatment was 6.24 ± 0.71 mm and on the left side was 6.26 ± 0.64 mm. After the treatment of hyponatremia, the mean ONSD on the right side was 5.81 ± 0.58 mm (95% 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, ($p < 0.001$). Thus, the study was well-powered to detect a change in ONSD and its correlation with serum sodium as the observed treatment effect was almost ten times the estimated treatment effect. The study also measured potential confounders like serum urea and blood glucose levels and these variables were found unchanged during the course of the ED stay. The mean duration of ED stay was 26.56 ± 9.57 hours. The observers measured ONSD at two different time points; at admission and discharge. More observations at different time points, say at 6th hourly intervals, and comparing with serum sodium levels at the same time points would have provided much more valuable data for further analysis. Some guidelines also suggests physicians consider measuring sodium 6th or 12th hourly during the correction.[10]

The authors also studied patient safety after discharge. Rapid correction of sodium can lead to ODS, especially in chronic hyponatremia. It can present as coma, locked-in syndrome, paresis, or dysarthria in the case of central pontine myelinolysis or as movement disorders like tremors, dystonia, and gait disorders in cases of extra pontine myelinolysis.[11] It can manifest from 1 to 14 days.[12] The authors have followed protocol for optimal correction of sodium. However, it is necessary to monitor at-risk patients (especially alcoholics) as 7% of cases still develop ODS despite a lower rate of correction.[10] The study could have benefitted from telephonic follow-up till a month after discharge from ED for monitoring these adverse events. Lack of readmission to ED is not an indicator of the absence of adverse events.

The strength of the study includes a novel concept, prospective design, well-defined inclusion criteria, and limited exclusion criteria making the results generalizable to Indian patients admitted to ED with symptoms of hyponatremia. Overall, the article provides valuable insights into the use of ONSD measurement in monitoring ICP and its potential as a guide for correcting hyponatremia in the ED. Serum sodium measurements are simple and non-expensive and it is unlikely that ONSD measurements would have replaced measurement of serum sodium even if the present study found a significant correlation. It would probably be used as an additional safety parameter along with serum sodium measurement to be assessed before discharge from ED. The study's limitations, such as the small sample size and the exclusion of patients with known or suspected raised ICP, are acknowledged.

Further research is needed to investigate the correlation between ONSD and sodium levels during hyponatremia correction, especially at multiple time points. Future trials comparing ONSD measurements using USG with a gold standard measurement such as CT during the correction of hyponatremia would also validate ONSD measurements. Researchers considering such trials should be mindful of the pitfalls of ONSD measurements and devise strategies to counter the inherent biases during ONSD measurements.

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