LETTER TO THE EDITOR

Optic Nerve Sheath Diameter in Hyponatremia: A Closer Look

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Dear Editor,

The research study by Uttanganakam et al.[1] brings to light a significant decrease in optic nerve sheath diameter (ONSD) after treatment of hyponatremia compared to pretreatment levels (mean right eye ONSD = 5.81 ± 0.58 mm [95% confidence interval (CI): 0.27–0.59, $p < 0.001$], and left eye ONSD = 5.79 ± 0.56 mm [95% CI: 0.29–0.64, $p < 0.001$] after hyponatremia correction; compared to mean right eye ONSD = 6.24 ± 0.71 mm and left eye ONSD = 6.26 ± 0.64 mm at presentation to the emergency department). However, the ONSD measurements failed to predict moderate to severe hyponatremia on receiver operating characteristic curve (ROC) analysis in the index prospective cohort. The author's attempt to extend the point of care utility of ultrasonographic measured ONSD to the emergency department deserves applause, though herewith we appraise a few facets to consider while extrapolating sub-millimetric variations in ONSD to clinical outcome.[2]

Uttanganakam et al. measured ONSD in the transverse plane with a high-frequency linear ultrasound probe using B-mode with the patient eyes closed, using hypoechoic lines behind the eye globe as reference. In B-scan, the ultrasonographic waves reach the optic nerve in a non-perpendicular array and hence measurements are predisposed to "scattering" and "blooming effect" artifact.[3] Despite the use of an ophthalmic setting Uttanganakam et al. contemplated, ONS wall brightness varies with 'gain' adjustments in B-scan. That said the index measurement being ONSD, variations in microns can affect the accuracy and precision of readings.[4] Therefore, we suggest that A-scan ultrasound (wherein the ONS is easily noticeable as hyperreflective spikes), be used especially so in the emergency department cohort of patients as time constraints and quicker decision-making are pertinent here.

That, 44.4% of cases in the index cohort had vomiting leaves the readers intrigued by the extent to which dehydration could have influenced ONSD measurements in hypovolemic hyponatremia. It is appreciated that the authors have carefully excluded hypervolemic hyponatremia, yet hypovolemia can alter cerebrospinal fluid production and dynamics consequently affecting ONSD measurements. Though Uttanganakam et al. 'practiced' ONSD imaging in 80 individual measurements prior to the study to avoid interobserver variability in measurements, how the authors 'blinded' their investigators to the nature of the image procured (before and after sodium correction) has been left unaccounted. Further, we appreciate that Uttanganakam et al. has carefully excluded head injury patients from their research wherein septations and ONS compartmentalization can mandate separate correlation of right and left ONSD measurements to the studied outcome, nevertheless, we believe that the average of either side ONSD can be better representative in the correlation and ROC analysis of the Uttanganakam et al. hyponatremia cohort of cases.[5]

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