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SHORT COMMUNICATION

Transorbital sonography and MRI reliability to assess optic nerve sheath diameter in idiopathic intracranial hypertension

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Abstract

Background and Purpose: The purpose of this study was to evaluate the performance of magnetic resonance imaging (MRI) in measuring the optic nerve sheath diameter (ONSD) compared to the established method transorbital sonography (TOS) in patients with idiopathic intracranial hypertension (IIH).

Methods: Twenty-three patients with IIH were prospectively included applying IIH diagnostic criteria. All patients received a lumbar puncture with assessment of the cerebrospinal fluid (CSF) opening pressure to assure the IIH diagnosis. Measurement of ONSD was performed 3 mm posterior to inner sclera surface in B-TOS by an expert examiner, while three independent neuroradiologists took measurements in axial T-weighted MRI examinations. The sella turcica with the pituitary gland (and potential presence of an empty sella) and the trigeminal cavity were also assessed on sagittal and transversal T1-weighted MRI images by one independent neuroradiologist.

Results: The means of ONSD between ultrasound and MRI measurements were 6.3 mm (standard deviation [SD] = 0.6 mm) and 6.2 mm (SD = 0.8 mm). The interrater reliability between three neuroradiologists showed a high interclass correlation coefficient (ICC) (confidence interval: $.573 < ICC < .8$; $p < .001$). In patients with an empty sella, the ONSD evaluated by MRI was 6.6 mm, while measuring 6.1 mm in patients without empty sella. No correlation between CSF opening pressure and ONSD was found.

Conclusions: MRI can reliably measure ONSD and yields similar results compared to TOS in patients with IIH. Moreover, patients with empty sella showed significantly larger ONSD than patients without empty sella.

KEYWORDS
idiopathic intracranial hypertension, MRI, ONSD, transorbital sonography

INTRODUCTION

The ongoing search for noninvasive methods to assess and monitor the intracranial pressure (ICP) in patients with idiopathic intracranial hypertension (IIH) is crucial, since the diagnosis of IIH is often protracted with patients having been constantly misdiagnosed with other headache syndromes prior to that. Patients with IIH suffer from a wide range of symptoms: from headaches and nausea to visual symptoms

such as transient obscurations of vision and diplopia. Delayed diagnosis with enduring elevation of ICP may lead to irreversible visual loss.[1]

In recent years, the evaluation of optic nerve sheath diameter (ONSD) has become widely used as a surrogate parameter for the non-invasive estimation of ICP.[2,3] Raised ICP can be detected by an increase in ONSD due to the continuity of meninges and subarachnoid space around the optic nerve.[2]

The advantages of transorbital sonography (TOS) include its fast completion, sensitivity for ONSD, and easy accessibility while having good sensitivity in estimating ICP indirectly and noninvasively.[2,3]

Magnetic resonance imaging (MRI) has also been used to evaluate indirect signs of increased ICP including an empty or partially empty sella turcica, optic nerve protrusion, distension of the optic nerve sheath (ONS), optic nerve tortuosity, slit-like ventricles, flattening of the posterior aspect of the optic globe, or transverse sinus stenosis.[4,5]

Up to date, only a few reports about reproducibility of ONSD measurements in patients with IIH using B-scan ultrasound compared to MRI have been published.[4,6]

The primary purpose of this study was to compare ONSD in patients with IIH by using TOS and MRI. Our second aim was to review the interrater reliability of ONSD measurement by MRI.

METHODS

Subjects

This study was performed according to the tenets of the Declaration of Helsinki and was approved by our institutional ethics committee (Number 160/16). Informed consent was obtained from each patient. All patients were recruited in the Department of Neurology of Saarland University Medical Center in Homburg/Saar, Germany. All patients were diagnosed with either primary or secondary IIH according to the current diagnostic criteria.

Each participant underwent general medical, ophthalmological, and neurological examination, basic laboratory investigations, and cerebral MRI. Lumbar puncture was performed in the left lateral recumbent position with posterior measurement of cerebrospinal fluid (CSF) opening pressure and legs fully extended. From January 2018 to December 2020, 23 patients with IIH were included (age 45 ± 13 years).[5]

All included patients received MRI and TOS on the same day. The lumbar puncture had been performed at least 7 days earlier.

Magnetic resonance imaging

All patients underwent cerebral MRI as a part of their treatment strategy. We used the routinely acquired 3-dimensional, T1-weighted magnetization-prepared rapid acquisition gradient echo in 3 or 1.5 Tesla scanners by Siemens (Skyra and Symphony Tim) without fat suppression and a slice thickness of 0.9 mm. This sequence offers great information gain for different neuroradiological questions without focusing on eyes and allows multiplanar reformations in high resolution due to small isotropic voxels. When assessing ONS), the absence of fat suppression is helpful due to good contrast between retroorbital fat hyperintensity and hypointense CSF surrounding optic nerve.

ONSD was defined for each eye 3 mm posterior to the inner sclera, measuring from outer wall to outer wall of the ONS and averaged for each patient.

The borders of ONS cannot be defined exactly by the means of this sequence so that they appear as a continuum between hypointensive CSF and hyperintensive fat. Considering that ocular ultrasonography measures the external diameter of the ONS, we divided the signal continuum that depicts the ONS into three equally sized regions and included the following illustration to the protocol, requiring that on both sides of the ONS two thirds of the thickness must be included in the horizontal diameter. Like in ocular ultrasonography, only the horizontal diameter was measured. It was crucial that the measurement was applied at the same distance behind the optic nerve papilla because the ONS is wider here than posteriorly. Some of the images were with contrast and others without contrast, which did not impact the assessment negatively.

Three independent neuroradiologists from our department, having been blinded to the results of ocular ultrasonography, evaluated obtained images according to the following protocol for each side: first, rotate the visualized data volume, performing axial and parasagittal reformation, adjusted to the optic nerve next to the globe. Second, mark the point in the optic nerve 3 mm behind the middle of sclera and make a coronal slice through this point (Figure 1B), and for third measure the ONS according to Figure 1A.

The measurements were performed using diagnostic monitors within the Sectra Picture Archiving and Communication System. This system enables simple multiplanar reformations by physicians and gives measurement tools allowing accurate evaluation of ONSD.

In addition, we assessed other radiological criteria for benign intracranial hypertension: pituitary and sella turcica measurements, the size of trigeminal cavity in axial slices, tortuosity of optic nerve, and the form of posterior globe wall. Other causes of intracranial hypertension were excluded.

Transorbital sonography

The trained sonographer involved in the measurements of ONSD was not involved in the management of the patients and performed the examinations according to previously described protocols.

Ultrasound-based ONSD measurement was performed using a portable ultrasound machine (My Lab Gold 30; Esaote, Genova, Italy) equipped with a linear transducer (LA 332, Esaote; 3–11 MHz) with a lateral resolution of 0.4 mm. The ONSD was defined using the distance between the external borders of the hyperechogenic area surrounding the optic nerve (Figure 1D), at the level of retina posterior to inner sclera surface. The optic nerve diameter was measured marking the internal borders of this formation (Figure 1D) as described in previous protocols.[7]

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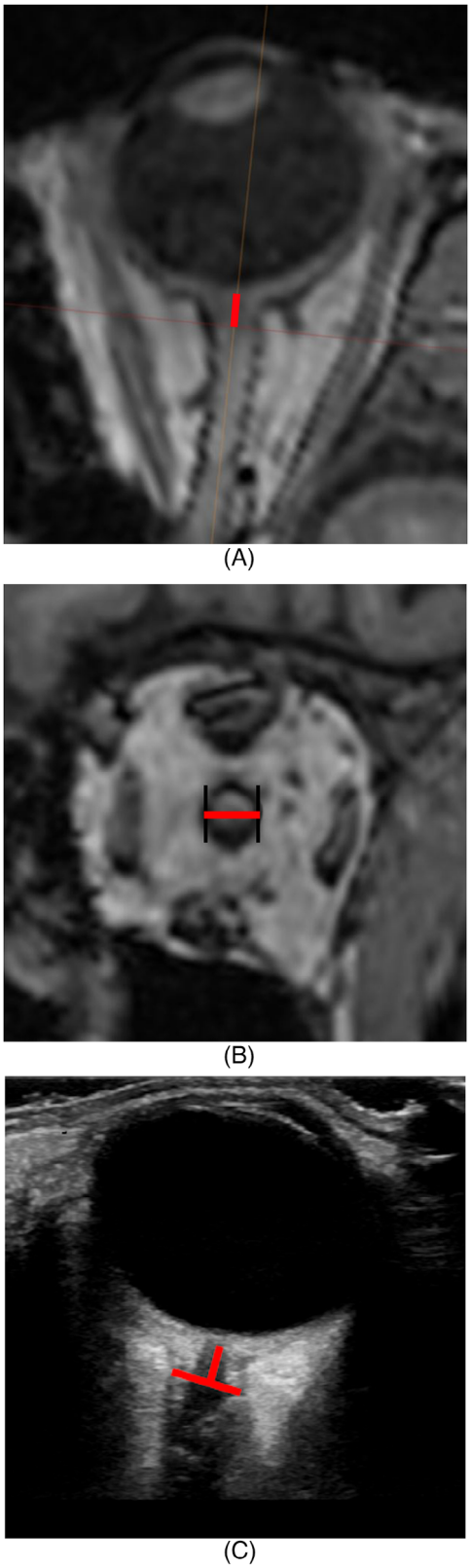

FIGURE 1 MRI and transorbital sonography methods. (A, B) Measurement of optic nerve sheath diameter from T2-weighted MRI: (A) Axial reformation to display the optic nerve. (B) Coronal 3 mm section posterior to the middle of sclera, used for measurement. (C) Measurement of optic nerve sheath diameter by ocular ultrasonography, measured 3 mm posterior to the papilla.

TABLE 1 Demographics and optic nerve sheath diameter measured with MRI or transorbital sonography in patients with IIH

Demographic data and ONSD of the IIH group ($n = 23$)	Patients with IIH
Age (years)	45.4 ± 13.1
Women (%)	70
Right ONSD TOS (mm)	6.3 ± 0.6
Left ONSD TOS (mm)	6.4 ± 0.6
Right ONSD MRI (mm) Rater 1	6.2 ± 0.8
Left ONSD MRI (mm) Rater 1	6.4 ± 0.6
Right ONSD MRI (mm) Rater 2	5.9 ± 0.8
Left ONSD MRI (mm) Rater 2	6.2 ± 0.9
Right ONSD MRI (mm) Rater 3	6.1 ± 0.9
Left ONSD MRI (mm) Rater 3	6.3 ± 0.9

Note: The data represent mean ± standard deviation unless otherwise indicated.
Abbreviations: IIH, idiopathic intracranial hypertension; *n*, number of subjects; OSND, optic nerve sheath diameter; TOS, transorbital sonography.

Statistical methods

We used linear regression analysis to investigate the correlation between the ONSD measured via TOS and via MRI in the left eye, the right eye, and the mean of both eyes. The same analysis was performed to assess the correlation between CSF opening pressure and ONSD measured by TOS and MRI. The interrater reliability between our three expert neuroradiologists was calculated performing an interclass correlation analysis and calculating an interclass correlation coefficient (ICC). In addition, we performed an unpaired *t*-test analysis to assess the differences of ONSD measurements between the groups "empty sella" and "partly-empty sella" compared to "no empty sella."

RESULTS

Demographics and ONSD measurement values are displayed in Table 1.

The linear regression of the mean ONSD of both eyes measured by three expert neuroradiologists in the images derived from MRI showed closed correlation to the mean ONSD measured by a neurosonology expert using TOS ($R = .68$, $p < .001$; Figure 2A). When each eye was assessed individually, we found the same close correlation between the ONSD measured in MRI and TOS (Figure 2B: right eye, $R = .55$, $p < .007$; Figure 2C: left eye, $R = .68$, $p < .001$).

To compare the ONSD values measured by three different expert neuroradiologists, we performed an interclass correlation analysis to assess whether ONSD could be reliably measured using MRI. This analysis yielded a highly significant ICC (confidence interval: $.573 < ICC < .8$; $p < .001$), indicating that measuring ONSD using MRI can be done in a reliable and reproducible way and is not strongly dependent on the examiner (Figure 3).

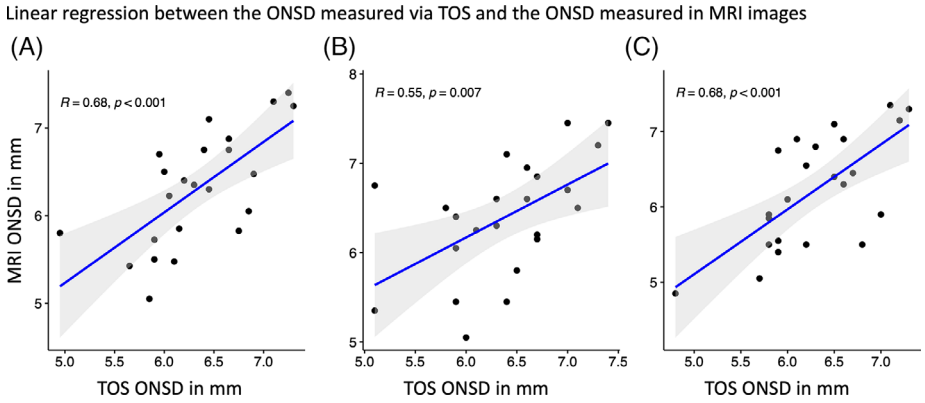

FIGURE 2 (A) Mean optic nerve sheath diameter (ONSD) of both eyes measured via transorbital sonography (TOS) compared to the mean ONSD of both eyes measured by three raters from MRI. (B) Mean ONSD of the right eye measured via TOS compared to the mean ONSD of the right eye measured by three raters from MRI. (C) Mean ONSD of the left eye measured via TOS compared to the mean ONSD of the left eye measured by three raters from MRI. Dots = each patient's mean ONSD; blue lines = regression lines; Gray shadows = 95% confidence intervals.

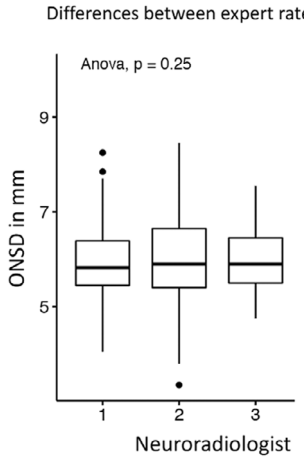

FIGURE 3 Differences of detected optic nerve sheath diameter (ONSD) values by the three expert raters. Dots = ONSD value outliers (measured by each rater); solid horizontal lines = first quartile, median, third quartile; boxes = interquartile range; whiskers = minimum or maximum, except for outliers.

Depending on the MRI findings, the patients were divided into three groups: no empty sella, partially empty sella, and completely empty sella. Patients with completely empty sella exhibited significantly higher ONSD values for the left and right eye, measured by MRI, and for the left eye, measured via TOS, than patients that showed no empty sella (TOS-measured ONSD left eye: $p = .02$; right eye: $p = .09$; MRI-measured ONSD left eye: $p = .03$; right eye: $p = .03$). In contrast, the size of trigeminal cavity did only correlate with the ONSD measured via TOS in the left eye ($R = .67$, $p = .003$). For the right eye ONSD measured via TOS and for the right and left eye ONSD measured via MRI, we found no correlation to the assessed size of the trigeminal cavity on the respective side (TOS ONSD right eye: $R = -.28$, $p = .303$; MRI ONSD right eye: $R = -.3$, $p = .26$; MRI ONSD left eye: $R = .12$, $p = .634$).

We did not find a correlation between CSF opening pressure and ONSD measured via TOS (left eye: $R = .08$, $p = .752$; right eye: $R = .03$, $p = .9$) or MRI (left eye: $R = .04$, $p = .88$; right eye: $R = .2$, $p = .43$).

DISCUSSION

The main finding of this study is that measuring the ONSD in IIH patients via MRI shows a similar accuracy like the established method TOS, which is demonstrated by the significant correlation between the values acquired by using these two methods. According to our knowledge, this is the first study that has measured ONSD in a reproducible way for adult patients with IIH, using TOS and MRI analysis performed by mutiple neuroradiologists. Moreover, we found a high ICC between three different expert neuroradiologists comparing the measurements of ONSD in MRI. Thus, it could be demonstrated that ONSD measurement using MRI can provide reproducible results independent of the examiner.

Previous studies have shown a good correlation between invasive ICP monitoring and TOS in measuring intracranial hypertension in cases of hydrocephalus, hepatic failure, and traumatic brain injury.[2,8–11] Furthermore, it was reported earlier, that also high-resolution MRI has been accurate in measuring ONSD.[12] MRI has the advantage of a high spatial resolution and a precise definition of orbital structure. Moreover, it can unveil other indirect signs of increased ICP, such as the empty sella.[13,14] In the present study, a slice thickness of 0.9 mm was used for the MRI images. This standard thickness was chosen to avoid a prolonged examination time in everyday clinical practice. Since ONSD may differ with as little as tenths of a millimeter with ICP changes, in retrospect it could be considered too thick. Nevertheless, at this resolutionwe still found a strong correlation between TOS- and MRI-measured ONSD values.

Previously, some authors described a good correlation between TOS and MRI in measuring ONSD in children with raised ICP.[15,16]

Similar to a previous study, in our study ONSD values in patients with an empty sella were significantly increased in both techniques compared to patients with a partially empty sella or without an empty sella.[6] This is an important observation taking into account that the empty sella is often used as an indirect sign of raised ICP in clinical practice. We did only find a correlation between the size of the left trigeminal cavity and the ONSD measured via TOS in the left eye,

suggesting that in our cohort the size of the trigeminal cavity was not an appropriate proxy for raised ICP, as all the other measures showed no significant correlation to the size of the trigeminal cavity. This might be explained by the fact that the trigeminal cavity is a 3-dimensional structure and thereby more complicated to be measured in a reproducible manner.

Discordant results exist concerning the correlation between ICP and ONSD. Some studies show a correlation between ONSD and CSF pressure, others do not.[17–19] In our study, it is difficult to establish any correlation because of the latency time between the lumbar puncture and the ONSD measurements. Furthermore, CSF pressure is constantly undergoing circadian variation. Apart from that, it has been postulated that long-lasting intracranial hypertension leads to a constant distension of the ONS and to a compartment syndrome in the ONS by fibrotic remodeling. Due to this, ONSD may become uncoupled of the opening pressure measured via lumbar puncture. Because of this, it is plausible why we did not find a correlation between the opening pressure in the lumbar puncture with the ONSD measured via MRI or TOS.[17,18]

This study has some limitations. First, the sample size is quite limited. Second, MRI- and TOS-based measurement of the ONSD is only a snapshot of the estimation of increased ICP and cannot replace the dynamic continuous invasive measurements of ICP but can just yield a clinical applicable proxy for ICP assessment. Moreover, the results could be biased by the ongoing treatment of the patients.

One must bear in mind that the TOS-measured ONSD remains an operator-dependent tool and therefore is only applicable in clinical practice with extensive previous training of the operator performing the examination. In our case, all examinations were performed by an experienced operator. The same applies, of course, to the ONSD measurement by MRI. Although we were able to show a good ICC, it was not near the excellent ICC for interrater reliability of TOS ONSD that has been demonstrated recently.[20] Since ONSD changes due to ICP changes may be very small, any method aiming to detect ICP changes by ONSD changes requires a minimal interrater variability.

In conclusion, we have found a good correlation between ONSD measured with TOS and MRI. The study has shown an agreement between these two methods, therefore both tools can be used as a noninvasive tool for detecting raised ICP with accuracy. Furthermore, we could show that measuring the same patients' ONSD with MRI performed by different experienced examiners yields coherent results, which demonstrates the methods' applicability for clinical practice.

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