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Optic Nerve Sheath Diameter is an Uncertain Indicator of a Transitory Ischemic Attack

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LETTER TO THE EDITOR

We read with interest the article by Kavak *et al*., on a retrospective study of the optic nerve sheath diameter (ONSD) in computed tomography to differentiate patients with transient ischemic attack (TIA) (n=77), acute ischemic stroke (AIS) (n=378), and healthy controls (n=318) [Kavak, R. P. *et al*., 2024]. The mean ONSD was significantly higher in both the AIS and TIA groups compared to controls and significantly higher in AIS subgroups than in TIA patients and controls [Kavak, R. P. *et al*., 2024]. In TIA patients ONSD was able to predict TIA with a sensitivity of 94.8%, and a specificity of 73.9% [Kavak, R. P. *et al*., 2024]. The study is impressive, but some points require further discussion.

The first point is the retrospective design of the study [Kavak, R. P. *et al*., 2024]. A retrospective design is unreliable for accurate measurement of morphological structures. A retrospective design also has the disadvantage that data can be missing, the accuracy of the data cannot be easily checked, desired missing or new data can no longer be generated, and clues for specific investigation are often not comprehensible. We should know how much data of the cohort was missing and to what extent this affected the results. How many patients were excluded due to missing data? In particular, a retrospective design implies that the measurement of ONSD was not standardised. Therefore, the slice in which the ONSD was measured may have changed from patient to patient and therefore may not be reproducible.

The second point is that if one assumes increased intracerebral pressure (ICP) as a cause of increased ONSD in TIA and AIS patients, the latency between the occurrence of TIA and AIS and the performance of CT should be included in the analysis. The ICP may decrease over time. In addition, due to physiologic diurnal rhythms, the ICP also has a high dynamic with highest values during the night [Finsterer, J. *et al*., 2023]. Therefore, it would have been imperative to carry out all the CT examinations at the same time of day. Since the study had a retrospective design, it is quite unlikely that these requirements were met.

The third point is that different causes of increased ICP have not been sufficiently ruled out. Increased ICP can occur not only in patients with TIA or AIS, but also in patients with hematoma, tumour, abscess, increased production of cerebrospinal fluid (CSF), choroid plexus tumour, decreased reabsorption of CSF, obstructive hydrocephalus, meningitis, increase in blood volume (e.g. increased cerebral blood flow during hypercarbia or aneurysms), venous stasis from venous sinus thromboses, elevated central venous pressures (e.g. heart failure), idiopathic or benign intracranial hypertension, skull deformities (e.g. craniosynostosis), hypervitaminosis A, or tetracycline use [Alves, C. A. P. F. *et al*., 2024]. We should know whether all of these differential causes of elevated ICP have been sufficiently ruled out before attributing the elevated ONSD to AIS or TIA.

A fourth point is that no explanation has been given as to why the ICP should increase during a TIA. Some TIAs only last a few seconds, so it is quite unlikely that an increase in ICP can develop in such a short time. Did ICP correlate with the duration of TIAs?

A fifth point is that group sizes were quite unequal, making statistical comparisons quite unreliable.

A sixth point is that the pathophysiology of AIS varies considerably and that the magnitude of the ICP increase may depend largely on the stroke mechanism. AIS due to macroangiopathy may affect ICP to a different extent than stroke due to venous sinus thrombosis.

The seventh point is that the interrater variability and the test-retest reliability were not assessed.

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In summary, the interesting study has limitations that put the results and their interpretation into perspective. Clarifying these weaknesses would strengthen the conclusions and could improve the study. Before attributing an ICP increase to AIS or TIA, all possible differential causes must be thoroughly ruled out. Furthermore, prospective multicentre studies are needed to assess whether the ONSD actually predicts TIA.

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