Commentary: Assessing the lamina cribrosa in idiopathic intracranial hypertension

The lamina cribrosa (LC) is a connective tissue mesh in the scleral canal of the optic nerve head that contains retinal ganglion cell axons and retinal blood vessels.[1] LC forms a barrier between 2 pressure compartments in the eye: intraocular pressure (IOP) in the intraocular space and cerebrospinal fluid pressure in the intraorbital subarachnoid space. A pressure difference between the intraocular and intracranial compartments at the site of the LC is called the translaminar pressure. This has been hypothesized to play a pathophysiological role in several optic nerve head diseases including papilledema. A change in either IOP or intracranial pressure may affect the homeostasis of the optic nerve head. LC has been known to undergo morphologic changes in papilledema.

Idiopathic intracranial hypertension (IIH) is characterized by elevated intracranial pressure of unclear cause manifesting with papilledema. Symptoms include headache, pulsatile tinnitus, transient visual obscurations, and diplopia from sixth nerve palsy. The basic hypothesis in IIH has been that increased translaminar pressure difference is detrimental to the axons of the optic nerve producing a mechanical insult and a disturbed axoplasmic transport. This leads to axoplasmic stasis, causing optic disc edema, eventually resulting in axonal death and visual loss.[2]

High-resolution techniques to study the accurate thickness of LC are both limited and difficult in human beings. Spectral domain-optical coherence tomography (SD-OCT) has been used to image the optic nerve head in papilledema. But a disadvantage of conventional OCT technology is that as depth increases, the resolution decreases, thus, imaging of deeper structures such as LC becomes challenging.

Enhanced depth imaging (EDI)-OCT was first reported in 2008 by Spaide *et al*.[3] to address the limitations of conventional SD-OCT for imaging deep ocular structures and to improve the image quality of deeper structures of the posterior pole.[4,5] This method involves placing the OCT apparatus close enough to the eye to create an inverted view of the fundus.[3,4] This places the coherence gate at a deeper plane than its usual position in the vitreous and moves the position of peak sensitivity from near the posterior vitreous in conventional OCT to the inner sclera for EDI-OCT. Using EDI-OCT, it is possible to visualize structures 500–800 μm deeper than with conventional OCT.

Another imaging tool is the swept-source optic coherence tomography (SS-OCT), also known as high-penetration OCT, that can enhance the visualization of deep ocular structures like the LC.[6,7] This uses longer wavelength light to study the deeper ocular structures.

There are not many studies that have used EDI-SD-OCT to study the LC in IIH. The present article is unique in that EDI-OCT was used to show several structural changes in LC from intracranial pressure alterations namely the depth, Bruch's membrane opening (BMO), and prelaminar tissue thickness. Undoubtedly their series is the largest in literature to elaborate on various morphologic changes of LC in IIH.[8]

Anterior displacement of the LC has been demonstrated using EDI-SD-OCT[9] and SS-OCT[7] in IIH compared with controls. Both studies suggest that increased intracranial pressure affecting the translaminar pressure creates anterior displacement of the LC. The present study has demonstrated 2 morphologic changes in support of this concept. The prelaminar tissue thickness measured as the area from the optic nerve head surface to the beginning of high reflectance of LC was found to be increased in IIH compared with controls. The anterior LC surface depth measured from the distance between the imaginary line passing through Bruch's membrane level and the anterior border of LC was shown to be reduced in IIH compared with controls. These features reflect the anterior displacement of the LC in IIH.

In contrast, EDI-OCT has shown posterior displacement of the LC in glaucoma.[9] The increased IOP compared with intracranial pressure affects the translaminar pressure causing the posterior displacement.

This study also demonstrated that BMO was significantly greater in IIH than the control group. Other studies using EDI-SD OCT have shown BMO was greater in papilledema patients with higher translaminar pressure difference. This reversed with reduction in that pressure.[10] Sibony *et al*. reported no statistically significant difference in BMO between the measurements before the treatment and after the treatment when the optic disc edema resolved, in their study of 23 IIH patients with optic disc edema.[11] However, the measurements were taken with SD-OCT, not with EDI-OCT.

No statistical difference was found in the LC thickness between IIH and healthy subjects in this study, just as demonstrated in another study using SS -OCT.[7] This favors the concept that translaminar pressure may play a more important role.

Overall this study implies that analysis of the morphologic changes of LC using EDI-OCT could be implemented as a valuable diagnostic and monitoring tool in intracranial hypertension.

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