Journal of Clinical & Experimental Ophthalmology

Laxmikanth and Girkin, J Clinic Experiment Ophthalmol 2011, S:4
DOI: 10.4172/2155-9570.S4-003

Research Article Open Access

Imaging Lamina Cribrosa with Spectral Domain Ocular Coherence Tomography: An overview

Laxmikanth Kankipati* and Christopher A. Girkin

University of Alabama at Birmingham, Ophthalmology, Birmingham, Alabama 35294, USA

Core Concepts

- Glaucomatous optic neuropathy is believed to start in the optic nerve head, the lamina cribrosa in particular, hence there is critical need for approach to imaging this region of the eye in-vivo.
- Understanding the complex interplay of intraocular pressure changes and lamina cribrosa is vital to our understanding of the disease progression.
- With newer imaging techniques, namely enhanced depth spectral domain ocular coherence tomography, it is now possible to image the anterior lamina and thus study its characteristics with respect to glaucomatous changes.
- There is a growing need for in vivo longitudinal studies that address the intraocular pressure-induced changes in the optic nerve head, in management of glaucoma.
- Imaging of the deep optic nerve and laminar surface may provide new endpoints to predict the development and progression of glaucoma.

Glaucoma is characterized by optic nerve head cupping and visual field defects that could be detected with long-term rigorous monitoring of patients. There is not yet a good screening test for the disease, hence emphasis should be on prevention of progression of the disease by risk reduction. Currently, the only proven method to prevent the progression glaucomatous disease is lowering the intraocular pressure (IOP). The precise mechanism by which IOP contributes to the development and progression of glaucoma is not completely understood [1,2]. This is, in part, due to the variability in individual susceptibility to IOP [3]. To better understand this individual susceptibility, it is essential to understand this relationship between IOP and glaucomatous optic neuropathy (GON) for effective management of the disease.

Clinically, the recognition of glaucomatous optic nerve damage is by the deepening and enlargement of the optic cup and thinning of the neuro-retinal rim [4]. The sclera is the main load-bearing tissue of the eye and deformations of the sclera due to the IOP changes are transmitted to the optic nerve head (ONH), the most susceptible part of the corneo-scleral shell. The cupping of the ONH in glaucoma, in most cases, is due to a combination of the two components – prelaminar and laminar cupping [5-8]. Prelaminar cupping of the ONH is characterized by progressive loss of the prelaminar tissues leading to increase in the depth and width of the optic cup (cup-disk ratio changes). Laminar cupping is a progressive posterior movement of the lamina cribrosa (LC) due to connective tissue remodeling that leads to progressive loss of the retinal ganglion cell (RGC) axons. The lamina is composed of a network of beams of connective tissue fibers that provide structural and functional support to the RGC axons as they pass through it into the optic nerve.

GON is believed to begin within the LC, changes include thinning, deformation, and compression of the connective tissue fibers and enlargement of pores [9,10]. Recent studies suggest that the trans-lamina cribrosa pressure difference (IOP vs. retrolaminar cerebrospinal fluid pressure around the optic nerve) is of importance in the pathophysiology of GON [10,11]. The altered biomechanical environment within the ONH and the LC may contribute to the disruption of the RGC axons and the subsequent loss of vision in GON. It is conventional belief that as IOP increases, the LC deforms posteriorly. Burgoyne et al. [12] developed ocular biomechanical models that show that as IOP changes, the LC and peripapillary sclera changes are much more complex and do not necessarily result in linear LC changes. Hence, there is a need for search for an association between changes in IOP and LC deformations [13].

The mainstay of clinical assessment of GON is by evaluation of the ONH by stereoscopic ophthalmoscopy. Newer imaging techniques have gained importance in recent years especially those with the ability to detect a longitudinal change in the optic disk. These include confocal scanning laser tomography, time domain optical coherence tomography, spectral domain ocular coherence tomography (SD-OCT) and scanning laser polarimetry. Post mortem studies in experimental glaucoma models of monkey eyes suggest that the earliest structural changes in ONH include a displacement of the LC, widening of scleral canal opening, and thickening of prelaminar tissue [5,6,13,15]. In vivo imaging of the ONH using high resolution imaging like the SD-OCT is thus essential to detect such early changes in these structures for better management of the disease.

OCT as a Measure of ONH changes

Optical Coherence Tomography (OCT) is a high-resolution, non-invasive imaging modality, first described by Huang et al [16], that generates an optical three dimensional section representative of the subsurface of the tissue being imaged, with a resolution approaching 10mm in the axial plane with the initial time-domain units [17]. Newer technological improvements include SD-OCT, where the reflected signal from tissues is captured by a spectrometer, thus eliminating the need for a moving reference mirror in the z-axis, greatly improving the resolution and imaging speed. In commercially available SD-OCT (Heidelberg engineering, Germany), the eye is illuminated with light from a broadband source. The scattered light from the tissue is combined with that from the reference arm to generate the interference signal. The anterior surface of the LC is well visualized on the SD-OCT, and this may be used in detecting glaucomatous changes (Figure 1).

***Corresponding author:** Laxmikanth Kankipati, Research associate, University of Alabama at Birmingham, Ophthalmology, Efh 4061530, 3rd Ave S, Birmingham, Alabama 35294,USA,12052970390, Fax: 12054880775; E-mail: klkanth@uab.edu

Received October 25, 2011; **Accepted** December 16, 2011; **Published** December 20, 2011

Citation: Laxmikanth K, Girkin CA (2011) Imaging Lamina Cribrosa with Spectral Domain Ocular Coherence Tomography: An overview. J Clinic Experiment Ophthalmol S4:003. doi:10.4172/2155-9570.S4-003

However, imaging of much deeper tissues with this technique is often limited due to the interference from more anterior tissues. Spaide et al [18], recently described an enhanced depth imaging (EDI) technique that improved the increased penetrance and axial resolution in choroid imaging. This EDI involved positioning of the SD-OCT device close enough to the eye to get an inverted image of the fundus, thus getting a better image of the deeper structures (Figure 2). Using this EDI SD-OCT, Lee et al. [19], visualized the LC with higher reflectivity and better contrast from the surrounding tissues, compared to that taken with conventional SD-OCT. Thus, in vivo imaging with EDI SD-OCT is currently the best commercially available technique available to image deeper ONH structures like the LC.

LCD changes to IOP alterations detected by SD-OCT

Agoumi et al. [1] utilized SD-OCT to determine the effect of transient elevation of IOP on laminar position. The authors transiently increased IOP in normal and glaucoma patients with an ophthalmodynamometer (Inami, Tokyo, Japan) and obtained SD-OCT images of the eye. They observed that there was no change in LC position for this transient raise of IOP but rather a compression of prelaminar tissues alone. This was in contrast to a displacement and/or change in thickness of LC, and expansion of the scleral canal observed in monkey and enucleated human eye models [10,14,20,21]. An advantage for the Agoumi et al study model was the ability to monitor real-time LC changes with changes in IOP, in vivo. However, one of the limitations to this study was the inability to measure the entire laminar surface, due in part to shadowing from blood vessels.

Also, even with EDI imaging, it is still difficult to visualize the full thickness of the LC, due to signal attenuation in the deep optic nerve and shadows cast by blood vessels. The latter issue may be partially resolved by using longer wavelength SD-OCT and/or post-processing imaging approaches. Recently, Girard et al. [22] have proposed an algorithm to enhance the contrast and removal of shadows in OCT images of the human ONH. They argue that by applying these simple compensation algorithms to remove blood vessel shadows, enhance contrast and improve tissue visibility at high depth facilitate the detection and segmentation of tissue boundaries to OCT images. This algorithm could be used both with the time-domain and spectral-domain OCT, and can also be used on existing images. However, for best contrast enhancement, prior shadow removal compensation step is necessary. Application of these techniques to EDI OCT images could provide us with better depth visualizations and would greatly enhance the segmentation and quantification of the ONH biomechanics.

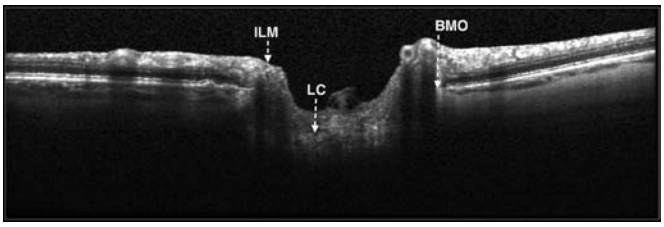

Figure 1: Radial section of OCT image (Heidelberg engineering, germany) of the optic nerve head in a normal adult human demonstrating the different structures of the ONH. ILM = Inner Limiting Membrane; BMO = Bruch's membrane opening; LC = Lamina Cribrosa.

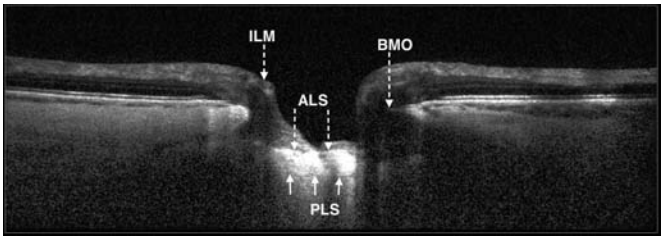

Figure 2: Enhanced depth spectral domain ocular coherence tomography (EDI SD-OCT) image of the ONH demonstrating better visualization of the anterior surface (ALS) and posterior surface (PLS, solid arrows) of the lamina cribrosa. ILM = Inner Limiting Membrane; BMO = Bruch's membrane opening.

Further studies are indicated to understand the complex interactions between IOP and ONH in the pathophysiology of GON. Understanding these interplays are essential for detection of the earliest clinically discernable ONH changes. This will likely provide new imaging targets to predict the development and progression of glaucoma.

References

1. Agoumi Y, Sharpe GP, Hutchison DM, Nicolela MT, Artes PH, et al. (2011) Laminar and prelaminar tissue displacement during intraocular pressure elevation in glaucoma patients and healthy controls. Ophthalmology 118: 52-59.

2. Downs JC, Roberts MD, Burgoyne CF, Hart RT (2009) Multiscale finite element modeling of the lamina cribrosa microarchitecture in the eye. Conf Proc IEEE Eng Med Biol Soc: 4277-4280.

3. Ian A Sigal, Michael D Roberts, Michael JA Girard, et al. (2010) Biomechanical changes of the optic disc. In: Levin LA, Albert DM ed. Ocular disease: Mechanisms and Management. Philadelphia Saunders/Elsevier Section 3, chapter 20: 153-164.

4. Morrison JC, Cepurna Ying Guo WO, Johnson EC (2011) Pathophysiology of human glaucomatous optic nerve damage: Insights from rodent models of glaucoma. Exp Eye Res 93: 156-164.

5. Yang H, Downs J C, Bellezza A, Thompson H, Burgoyne C F, (2007) 3-D histomorphometry of the normal and early glaucomatous monkey optic nerve head: prelaminar neural tissues and cupping. Invest Ophthalmol. Vis Sci 48: 5068-5084.

6. Yang H, Downs J C, Girkin C, Sakata L, Bellezza A, et al. (2007b) 3-D histomorphometry of the normal and early glaucomatous monkey optic nerve head: lamina cribrosa and peripapillary scleral position and thickness. Invest Ophthalmol Vis Sci 48: 4597-4607.

7. Burgoyne CF, Downs JC (2008) Premise and prediction-how optic nerve head biomechanics underlies the susceptibility and clinical behavior of the aged optic nerve head. J Glaucoma 17: 318-328.

8. Crawford Downs J, Roberts MD, Sigal IA (2011) Glaucomatous cupping of the lamina cribrosa: A review of the evidence for active progressive remodeling as a mechanism. Exp Eye Res 93: 133-140.

9. Inoue R, Hangai M, Kotera Y, Nakanishi H, Mori S, et al. (2009) Three-dimensional high-speed optical coherence tomography imaging of lamina cribrosa in glaucoma. Ophthalmology Feb 116: 214-222.

10. Jonas JB, Jonas RA, Jonas SB, Panda-Jonas S (2011) Lamina cribrosa thickness correlated with peripapillary sclera thickness. Acta Ophthalmol [Epub ahead of print].

11. Ren R, Wang N, Zhang X, Cui T, Jonas JB (2011) Trans-lamina cribrosa pressure difference correlated with neuroretinal rim area in glaucoma. Graefes Arch Clin Exp Ophthalmol 249: 1057-1063.

12. Burgoyne CF, Downs JC, Bellezza AJ, Hart RT (2004) Three-Dimensional Reconstruction of Normal and Early Glaucoma Monkey Optic Nerve Head Connective Tissues. Invest Ophthalmol Vis Sci 45: 4388-4399.

13. Sigal IA, Yang H, Roberts MD, Burgoyne CF, Downs JC (2011) IOP-induced lamina cribrosa displacement and scleral canal expansion: an analysis of factor interactions using parameterized eye-specific models. Invest Ophthalmol Vis Sci 52: 1896-1907.

14. Strouthidis NG, Fortune B, Yang H, Sigal IA, Burgoyne CF (2011) Longitudinal change detected by spectral domain optical coherence tomography in the optic nerve head and peripapillary retina in experimental glaucoma. Invest Ophthalmol Vis Sci 52: 1206-1219.

15. Downs JC, Yang H, Girkin C, Sakata L, Bellezza A, et al. (2007) Three-Dimensional Histomorphometry of the Normal and Early Glaucomatous Monkey Optic Nerve Head: Neural Canal and Subarachnoid Space Architecture. Invest Ophthalmol Vis Sci 48: 3195-3208.

16. Huang D, Swanson EA, Lin CP, Schuman JS, Stinson WG, et al. (1991) Optical coherence tomography. Science 254: 1178-1181.

17. Fatehee N, Yu PK, Morgan WH, Cringle SJ, Yu DY (2011) Correlating morphometric parameters of the porcine optic nerve head in spectral domain optical coherence tomography with histological sections. Br J Ophthalmol 95: 585-589.

18. Spaide RF, Koizumi H, Pozzoni MC (2008) Enhanced Depth Imaging Spectral-Domain Optical Coherence Tomography. Am J Ophthalmol 146: 496-500.

19. Lee EJ, Kim TW, Weinreb RN, Park KH, Kim SH, et al. (2011) Visualization of the Lamina Cribrosa Using Enhanced Depth Imaging Spectral-Domain Optical Coherence Tomography. Am J Ophthalmol 152: 87-95.

20. Bellezza AJ, Rintalan CJ, Thompson HW, Downs JC, Hart RT, et al. (2003) Deformation of the Lamina Cribrosa and Anterior Scleral Canal Wall in Early Experimental Glaucoma. Invest Ophthalmol Vis Sci 44: 623-637.

21. Roberts MD, Sigal IA, Liang Y, Burgoyne CF, Downs JC (2010) Changes in the biomechanical response of the optic nerve head in early experimental glaucoma. Invest Ophthalmol Vis Sci 51: 5675-5684.

22. Girard MJ, Strouthidis NG, Ethier CR, Mari JM (2011) Shadow removal and contrast enhancement in optical coherence tomography images of the human optic nerve head. Invest Ophthalmol Vis Sci 52: 7738-7748.

This article was originally published in a special issue, **Glaucoma** handled by Editor(s). Dr. Zaher Sbeity, University of Bonn, Germany