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Ocular Toxoplasmosis Presenting With Focal Retinal Nerve Fiber Atrophy Simulating Glaucoma

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Abstract

Purpose—We describe a patient with ocular hypertension and non-glaucomatous retinal nerve fiber layer (RNFL) atrophy associated with an ocular toxoplasmosis retinal lesion.

Patient and Method—Single case report

Results—An RNFL defect was identified adjacent to a circumscribed pigmented chorioretinal lesion superior to the macular region. The optic disc showed a prominent central cup without focal neural rim atrophy. Red-free photography and RNFL imaging using optical coherence tomography and scanning laser polarimetry showed corresponding focal RNFL atrophy. The toxoplasmosis lesion was characterized by an apparent full thickness disruption in retinal architecture, including the retinal ganglion cell layer, using spectral-domain OCT.

Conclusions—Non-glaucomatous retinal lesions may simulate glaucomatous RNFL atrophy and visual field loss, particularly when focal damage to the retinal ganglion cells occurs. Careful inspection of the retina and, optic disc with attention to the integrity of the neural rim, and analysis of the pattern of RNFL loss in proximity to a retinal lesion, may enable the clinician to differentiate glaucomatous and non-glaucomatous pathogenic mechanisms.

Ocular toxoplasmosis, caused by the obligate intracellular parasite *Toxoplasma gondii*, may produce lesions that cause visual field defects corresponding to disruption of the retinal nerve fiber layer (RNFL), typically with lesions proximal to the disc producing larger visual field defects¹. Recently, optical coherence tomography (OCT) imaging has better characterized the associated anatomic changes including retinal thinning, macular edema, vitreomacular traction and macular schisis² which occur during the inflammatory phase of the disease. Herein we describe a patient with ocular hypertension presenting with focal RNFL atrophy and standard visual field abnormalities that simulated glaucoma, but were attributed to a toxoplasmosis retinal lesion in which the disruption in retinal architecture was characterized by spectral-domain OCT.

A 44 year-old female presented for evaluation of glaucoma in the left eye. The baseline untreated intraocular pressure was 25 mmHg and ocular hypotensive medication was initiated by the referring physician using latanoprost 0.005% once daily in the evening. Examination of the left eye demonstrated a visual acuity of 20/25, central corneal thickness of 568 μ m, and the intraocular pressure measured 18 mmHg. Standard automated perimetry revealed an inferior scotoma close to fixation, and fundusoscopic examination revealed a corresponding wedge-

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shaped superior RNFL defect and adjacent circumscribed pigmented chorioretinal lesion superior to the macular region, consistent with toxoplasmosis (Figure 1). The optic disc showed a prominent central cup without focal neural rim atrophy. As illustrated in Figure 2, topography measurements generated using confocal scanning laser ophthalmoscopy (HRT, Heidelberg Engineering, GmbH, Heidelberg, Germany) revealed an abnormal global Moorfield regression analysis classification and normal optic disc area. RNFL imaging using Stratus™ OCT (Carl Zeiss Meditec, Dublin, CA) and scanning laser polarimetry (GDx-ECC, Carl Zeiss Meditec, Dublin, CA) demonstrated focal RNFL thinning within the superior arcuate region and adjacent to the retinal lesion. Cross-sectional OCT images were obtained through the toxoplasmosis scar (Figure 3) using spectral-domain OCT (RTVue 100, Optovue, Fremont, CA) and revealed disruption of all retinal layers including the retinal ganglion cell layer, and cross-sectional imaging nasal to the lesion demonstrated focal RNFL atrophy.

The glaucomas represent a group of diseases that result in progressive retinal ganglion cell death, producing a specific pattern of RNFL atrophy, optic nerve head and visual field damage. In this particular patient, these observations were associated with elevated intraocular pressure. This case demonstrates that non-glaucomatous lesions may produce similar patterns of RNFL atrophy and visual field loss which may be identified clinically and quantified using advanced imaging technology. Similar clinical observations were established by Knighton and colleagues who observed RNFL defects using red-free photography following iatrogenic retinal laser photocoagulation³. In our patient, spectral domain OCT technology nicely demonstrated disruption in all layers of retinal architecture resulting in localized atrophy of the ganglion cells and their axons. In contrast to glaucomatous optic neuropathy which often produces focal thinning or notching of the neural rim adjacent to focal RNFL atrophy, the integrity of the neural rim in this patient was relatively well preserved.

In summary, this case emphasizes that non-glaucomatous retinal lesions may simulate glaucoma, particularly when focal damage to the retinal ganglion cells occur. Our patient developed these manifestations in the setting of ocular hypertension further complicating the clinical interpretation of the lesions. Careful inspection of the retina and optic disc with attention to the integrity of the neural rim, and analysis of the pattern of RNFL loss in proximity to the retinal lesion, may enable the clinician to differentiate glaucomatous from non-glaucomatous pathogenic mechanisms. Ocular imaging technology may facilitate the assessment and quantification of RNFL atrophy in such eyes. As with perimetry, clinicians should avoid interpretation of imaging studies in isolation of other clinically relevant findings.

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Figure 1.

Optic disc (top left) and standard automated perimetry (top right) of the left eye demonstrate absence of neural rim thinning or notching, and inferior scotoma near fixation. Fundus and red-free photograph (bottom left and right) demonstrate superior retinal nerve fiber layer atrophy (RNFL, white arrows) adjacent to a pigmented chorioretinal scar above the macula, consistent with toxoplasmosis.

Figure 2.

Heidelberg retina tomograph (HRT, top) demonstrates a normal disc area (2.680mm²) and abnormal global Moorfield regression analysis classification. RNFL assessment using scanning laser polarimetry with enhanced corneal compensation (GDx-ECC, middle) and StratusTM optical coherence tomography (OCT, bottom) illustrate focal reduction in supero-temporal region.

Figure 3. Cross-sectional spectral-domain OCT image through the toxoplasmosis lesion illustrates diffuse disruption in retinal architecture involving all retinal layers including the ganglion cell layer. Cross-sectional image nasal to the lesion demonstrates focal RNFL atrophy (white arrows).