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Non-Arteritic Anterior Ischemic Optic Neuropathy after YAG Capsulotomy - A Case Report

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Abstract

A 61 year old African American female underwent a Yttrium-Aluminum-Garnett (YAG) laser capsulotomy in the left eye. She presented with pain and blurry vision one day later and was found to have elevated intraocular pressure which persisted for a few days in spite of topical eye pressure lowering eye drops and oral acetazolamide. One week later the patient developed optic disc edema and a new visual field defect in the left eye. Local ocular and systemic evaluation was unremarkable for other causes of optic neuropathy, and the patient developed optic disc pallor several weeks later. The presumptive diagnosis was non-arteritic anterior ischemic optic neuropathy which has been previously reported after cataract surgery although occurrence after YAG capsulotomy is not known.

Keywords: YAG laser capsulotomy; Non-arteritic anterior; Ischemic optic neuropathy

Introduction

This report details a single patient who patient who underwent a YAG laser capsulotomy and developed prolonged elevation of intraocular pressure for several days. The patient developed optic disc edema and a visual field defect a week later and this author proposes that the prolonged intraocular pressure elevation precipitated a non-arteritric anterior ischemic optic neuropathy but not glaucomatous optic neuropathy.

Materials and Methods

This article represents a signal patient case report of a patient who developed a non-arteritic anterior ischemic optic neuropathy subsequent to prolonged intraocular pressure elevation after a YAG laser capsulotomy. Literature review of YAG laser capsulotomy complications and cases of NAION after cataract surgery were reviewed to help gain insight into the mechanism of injury in this case.

A 61 year old African American underwent Yttrium-Aluminum-Garnett (YAG) laser capsulotomy in the left eye twenty years after cataract surgery for a traumatic cataract. The procedure note from the referring Ophthalmologist detailed pre-procedure acuity of 20/25 and intraocular pressure (IOP) of 15 mm Hg. The patient's YAG capsulotomy procedure involved 69 bursts of 3.0-4.0 mj/burst for a total of at least 207 mj of energy. One drop of apraclonidine was placed in the left eye after the procedure and IOP was recorded at 25 mm Hg 60 min later.

History

The patient reported pain and blurry vision in her procedural eye one day later. Visual acuity was 20/60 and IOP was 46 mm Hg. She was treated with topical IOP lowering drops (Brimonidine, Dorzolamide-Timolol, Latanoprost) as well as one oral acetazolamide 500 mg sequel and asked to return one day later. On post-procedure day 2, the acuity had improved to 20/40, but IOP was 61 mm Hg. The treating Ophthalmologist performed an anterior chamber paracentesis which immediately lowered the IOP to 15 mm Hg. Over the next few days, the patient was continued on topical IOP drops and started on topical prednisolone acetate for associated anterior chamber cells. At day 4 post procedure, anterior vitreous cells were noted and on day 7, optic disc edema was visualized. A visual field study was performed which showed a superonasal defect (Figure 1). The patient was then referred to the Medical Retina clinic for additional evaluation.

Examination

Evaluation two weeks post procedure identified visual acuity of 20/30, IOP 16 mm Hg, a trace afferent pupillary defect in the left eye. Superonasal visual field defect was confirmed on confrontation testing. Slit lamp exam showed mild conjunctival injection, a clear cornea with fine keratic precipitates and 1+ anterior chamber cell. The iris was flat with rubeosis or nodules. The sulcus intraocular lens (IOL) was centered without posterior dislocation and there was a capsular opacity noted in the residual capsule complex. Dilated exam revealed 1+ anterior vitreous cell, optic disc edema (Figure 2) and one small blot intra-retinal hemorrhage inferiorly (not seen on photo). Retinal nerve fiber layer optical coherence tomography (RNFL-OCT) demonstrated thickening of the left eye (Figure 3). The ganglion cell analysis was normal bilaterally (Figure 4). OCT of the macula did not reveal any cystoid edema. Fluorescein angiography showed normal transit in the left eye without patchy choroidal filling, vascular occlusions, vasculitis, nor cystoid macular edema. Significant optic disc fluorescence, however, was noted in the left eye (Figure 5).

Evaluation

A broad differential diagnosis of vision loss after YAG capsulotomy was considered and encompassed several categories including direct YAG laser induced injury, infectious, inflammatory, vascular occlusive and IOP related causes. Direct laser induced damage could cause corneal edema or endothelial cell injury, pits/pockmarks in the IOL and dislocation of the IOL from a YAG capsulotomy which is too large. Infectious etiologies included delayed/sequestered endophthalmitis from *Propionibacterium acnes* or *Diphtheroid* sp., as well as unmasked syphilis or tuberculosis. Non-infectious uveitis including Sarcoidosis or Wegener's Granulomatosis could have been exacerbated by the inflammation associated with YAG capsulotomy. Occlusive vascular disease caused by YAG capsulotomy

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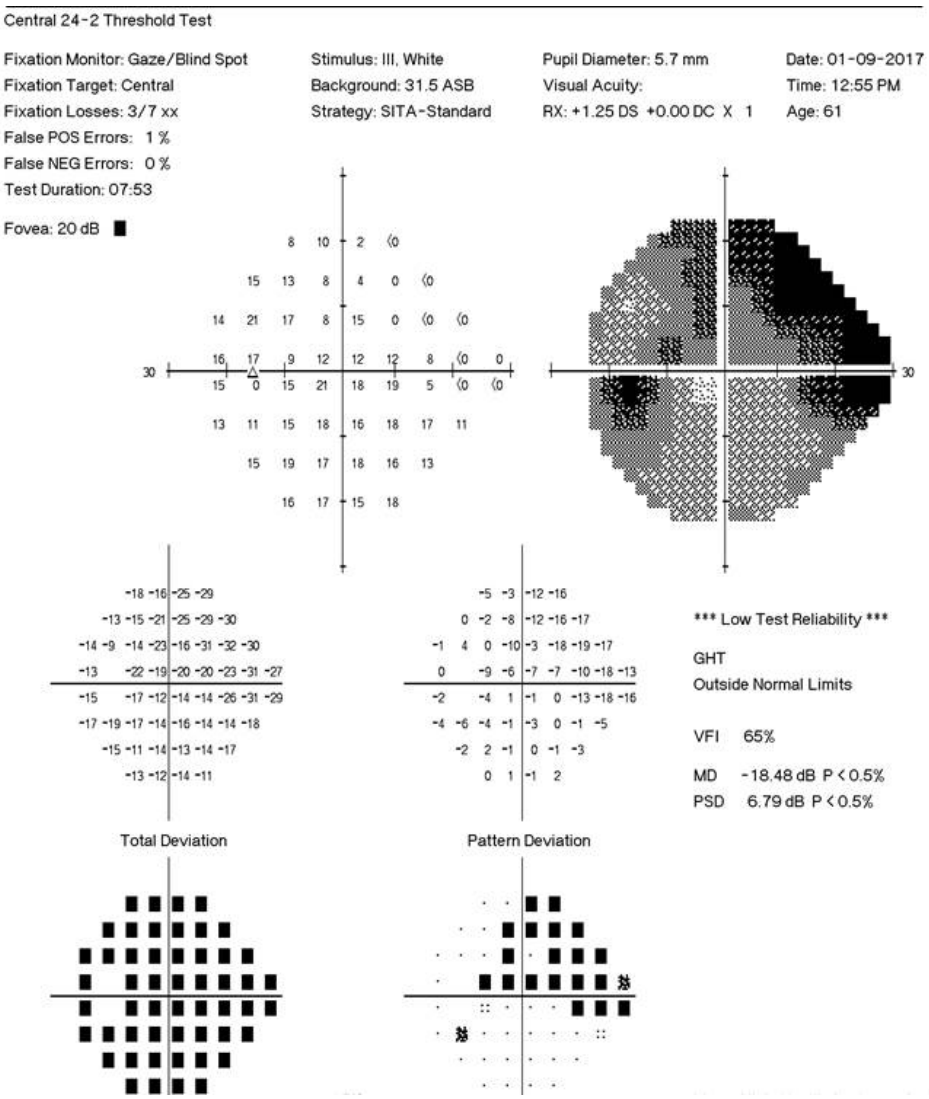

Figure 1: Humphrey visual field 24-2 of the left eye demonstrating a superonasal defect.

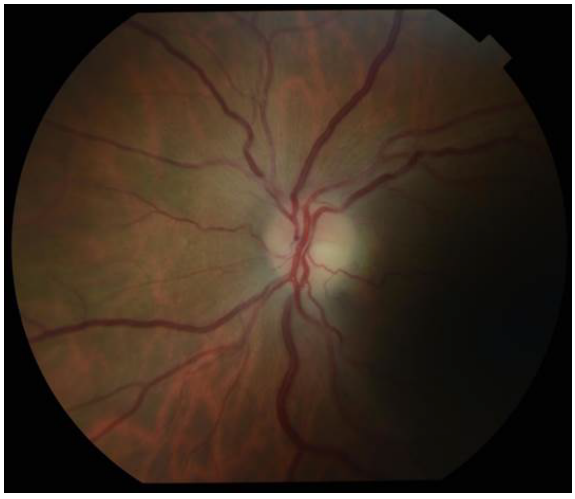

Figure 2: Fundus photo showing optic disc edema in the left eye.

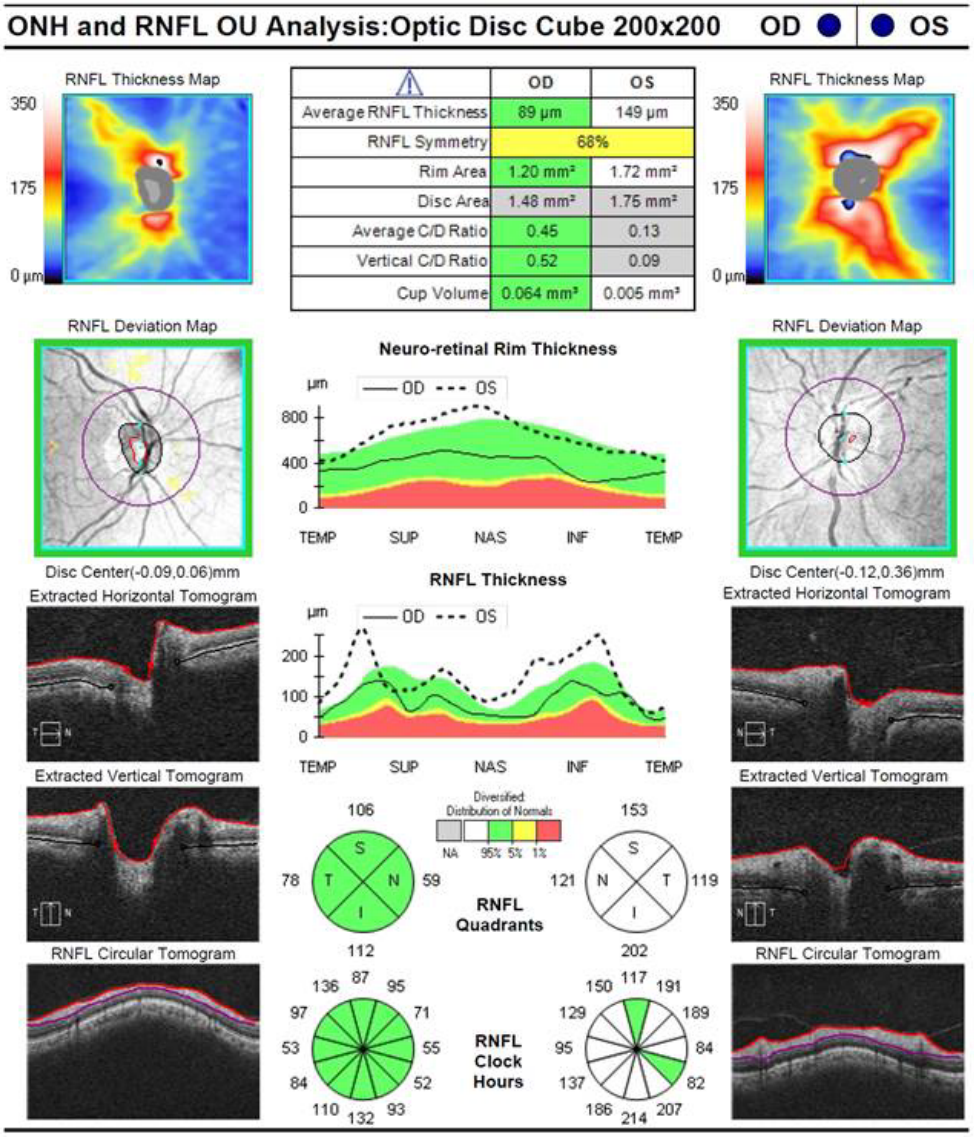

Figure 3: Retinal nerve fiber layer optical coherence tomgraphy (RNFL-OCT) revealing thickening of the nerve fiber layer in the left eye.

affecting the retinal vessels or choroid was considered. Finally, IOP induced damage directly or via associated inflammation causing anterior ischemic optic neuropathy was considered.

In order to rule out significant vision threatening etiologies of vision loss, laboratory studies were performed which showed mild elevation of inflammatory markers, but no serologic evidence of Sarcoidosis, Wegener's Granulomatosis, Tuberculosis or Syphilis. A vitreous tap was performed and was negative for growth of aerobes or anaerobes at one week, ruling out delayed or sequestered endophthalmitis. Magnetic Resonance Imaging (MRI) of the head failed to reveal any optic nerve or orbital lesions and there were no masses or signs of obstructive hydrocephalus or cerebral sinus thromboses seen on Magnetic Resonance Venography (MRV). The patient was started on oral prednisone and underwent a temporal artery biopsy on the left side which did not reveal any granulomatous inflammation suggestive of giant cell arteritis. Topical prednisolone acetate was started for the mild anterior chamber and anterior vitreous cells.

Clinical course

Over the next several weeks, the patient had improvement in the mild uveitis on the topical corticosteroid as well as resolution of the optic disc edema which progressed to inferior disc pallor (Figure 6). Repeat visual field study showed stabilization of the superior acute defect (Figure 7) subsequent thinning of the RNFL (Figure 8) as well as the Ganglion cell layers (Figure 9).

Discussion

The patient in this report appeared to develop a non-arteritic anterior ischemic optic neuropathy (NAION) secondary to prolonged IOP elevation after a YAG capsulotomy in the left eye. NAION has been previously reported after uncomplicated cataract surgery at rates estimated at one case per every 1000 to 2000 cataract surgeries [1-4] although reports conflict as to whether this is higher than the spontaneous rate in age-matched adults [4]. The occurrence of NAION after YAG capsulotomy is unknown.

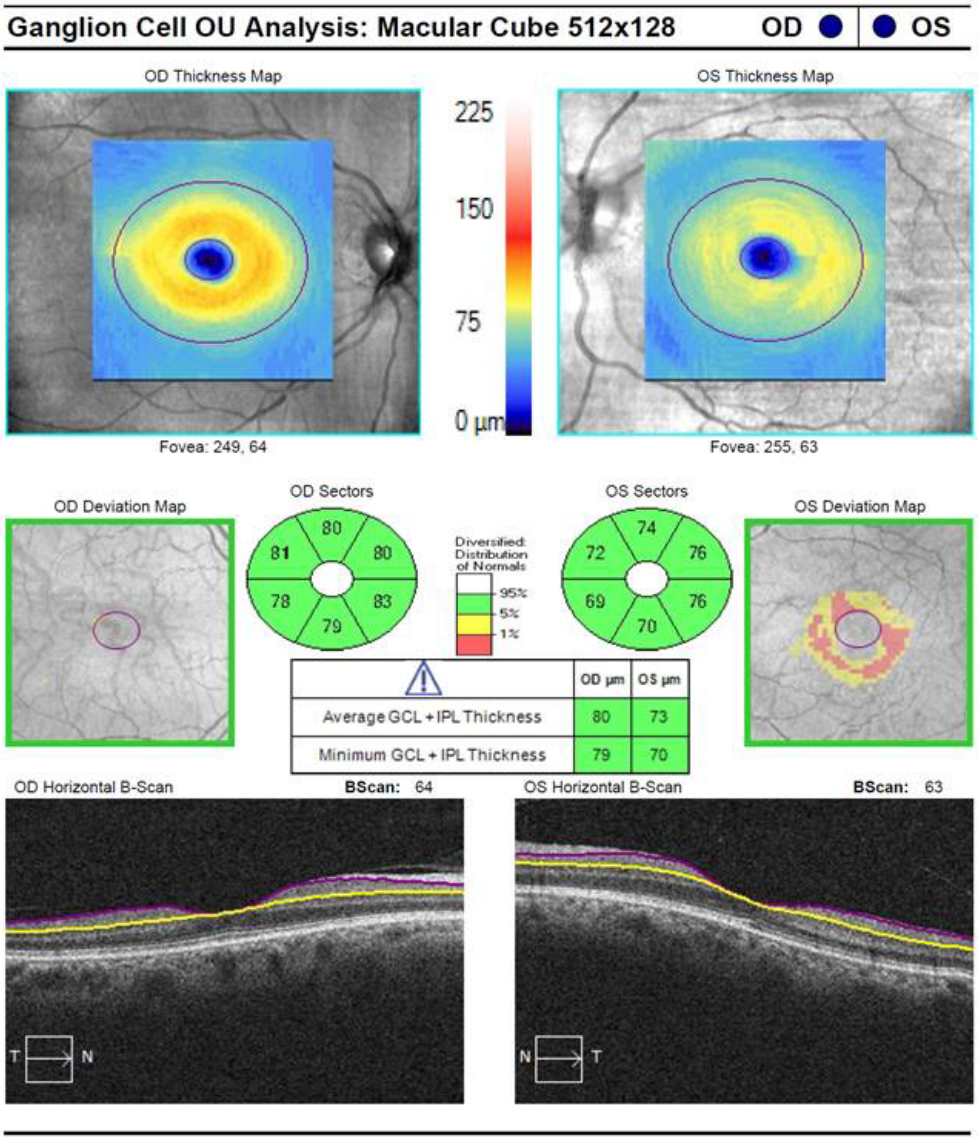

Figure 4: Ganglion cell analysis showing normal thickness bilaterally.

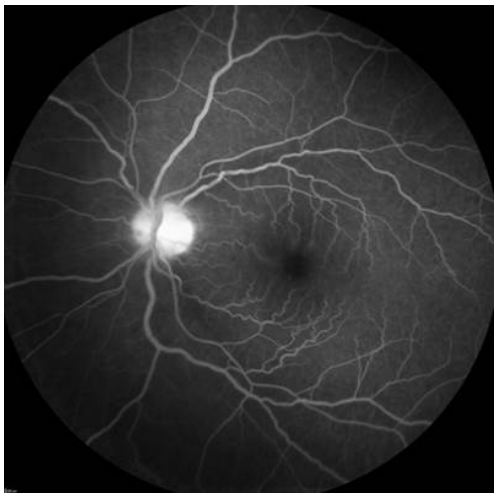

Figure 5: Fluorescein angiography confirming optic disc edema of the left eye.

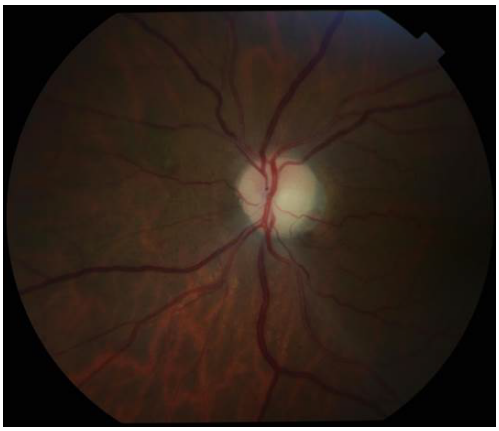

Figure 6: Subsequent fundus photograph demonstrating inferior optic disc pallor in the left eye.

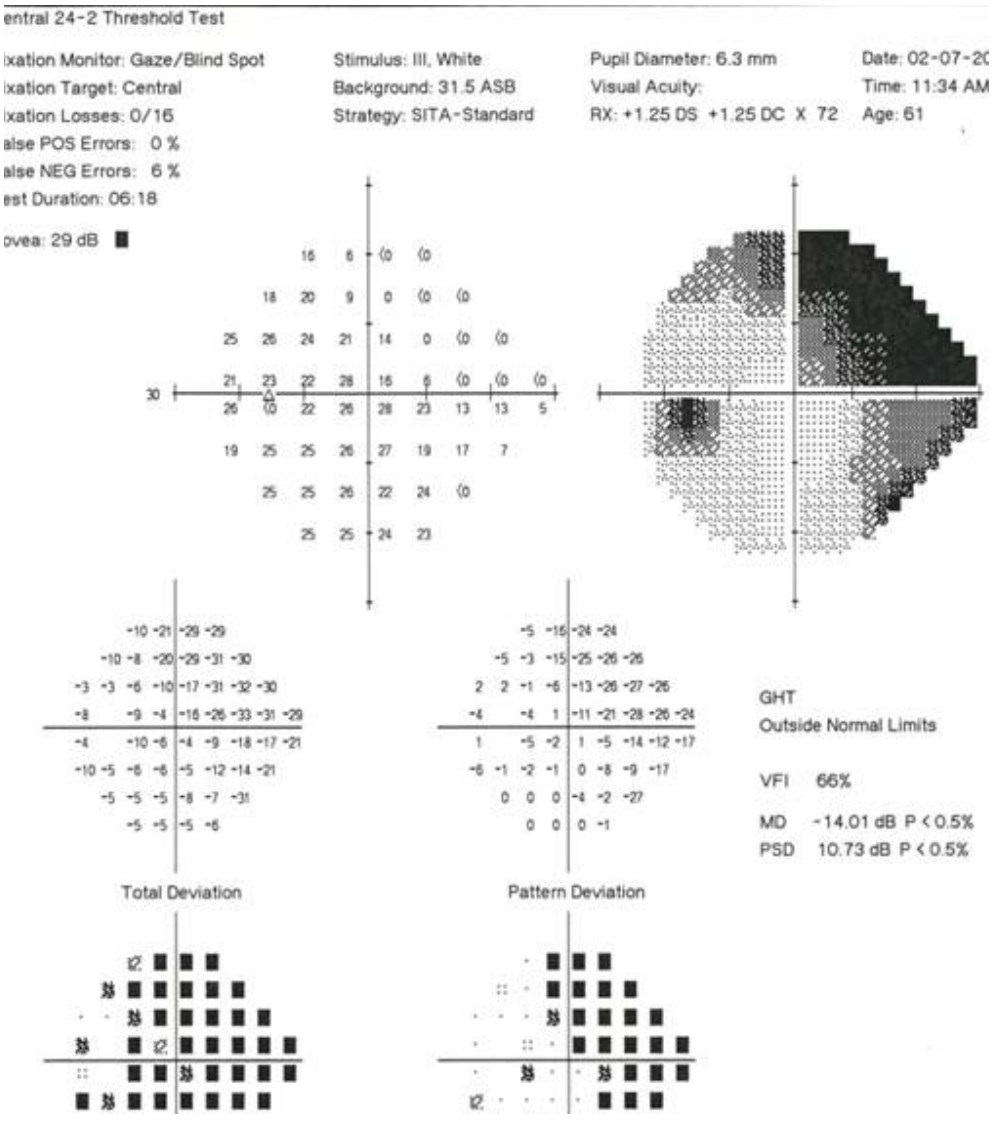

Figure 7: Repeat 24-2 visual field study showing persistence but stabilization of the superonasal defect.

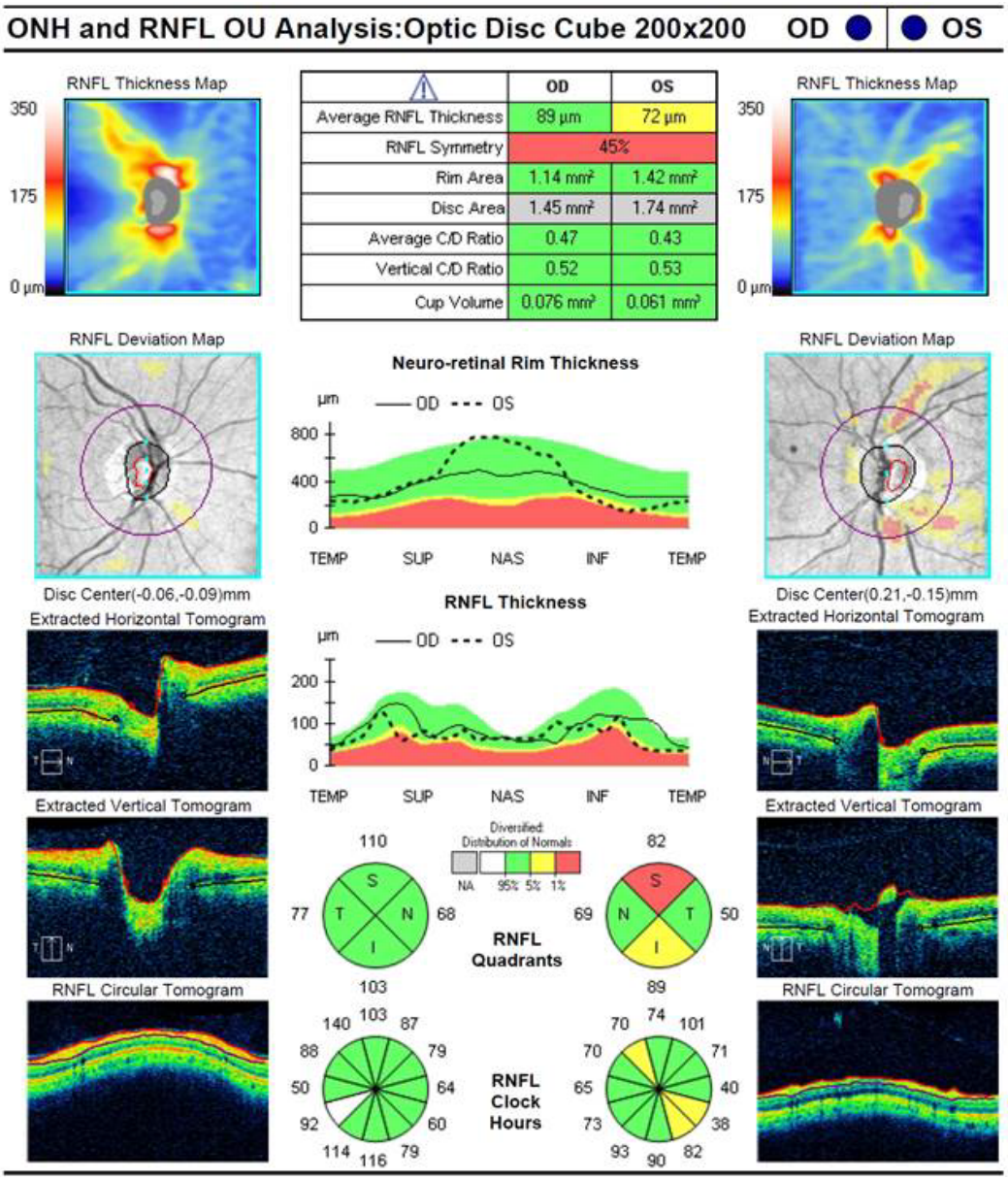

Figure 8: Subsequent RNFL-OCT showing thinning of the retinal nerve fiber layer of the left eye.

The mechanism by which most cases of NAION occur is likely secondary to reduced optic nerve perfusion secondary to nocturnal hypotension in an eye with a small cup (i.e., disc at risk) [3]. Classically this condition presents with painless vision loss in a middle aged adult with optic disc edema and a corresponding visual field defect. Non-arteritic anterior ischemic optic neuropathy a few hours to days after a cataract surgery has been proposed to be related to elevated intraocular pressure without reductions in optic nerve perfusion in the intraoperative or post-operative period [1-4]. A delayed variant of anterior ischemic optic neuropathy occurring weeks to months after cataract surgery has been reported to occur but its mechanism is less understood.

A growing body of Glaucoma [5] literature suggests that ocular perfusion pressure between to affected by both systolic blood pressure and intraocular pressure as shown by the equation.

(Ocular Perfusion Pressure=Systolic Blood Pressure - Intraocular Pressure)

In this patient's case, the large amount of YAG laser energy used during the capsulotomy procedure likely induced significant inflammation which elevated the intraocular pressure enough to reduce the overall ocular perfusion pressure leading to an NAION. While most cases of non-arteritic anterior ischemic optic neuropathy typically present without pain, the significant IOP elevation and coincident inflammation was likely the cause of the painful vision loss.

This is the first known reported case of an NAION after YAG Capsulotomy. There is a case report of capsular block glaucoma from retained viscoelastic in the capsular bag and associated elevated IOP induced damage causing an anterior ischemic optic neuropathy [6]. In that report, the YAG capsulotomy relieved the viscoelastic induced

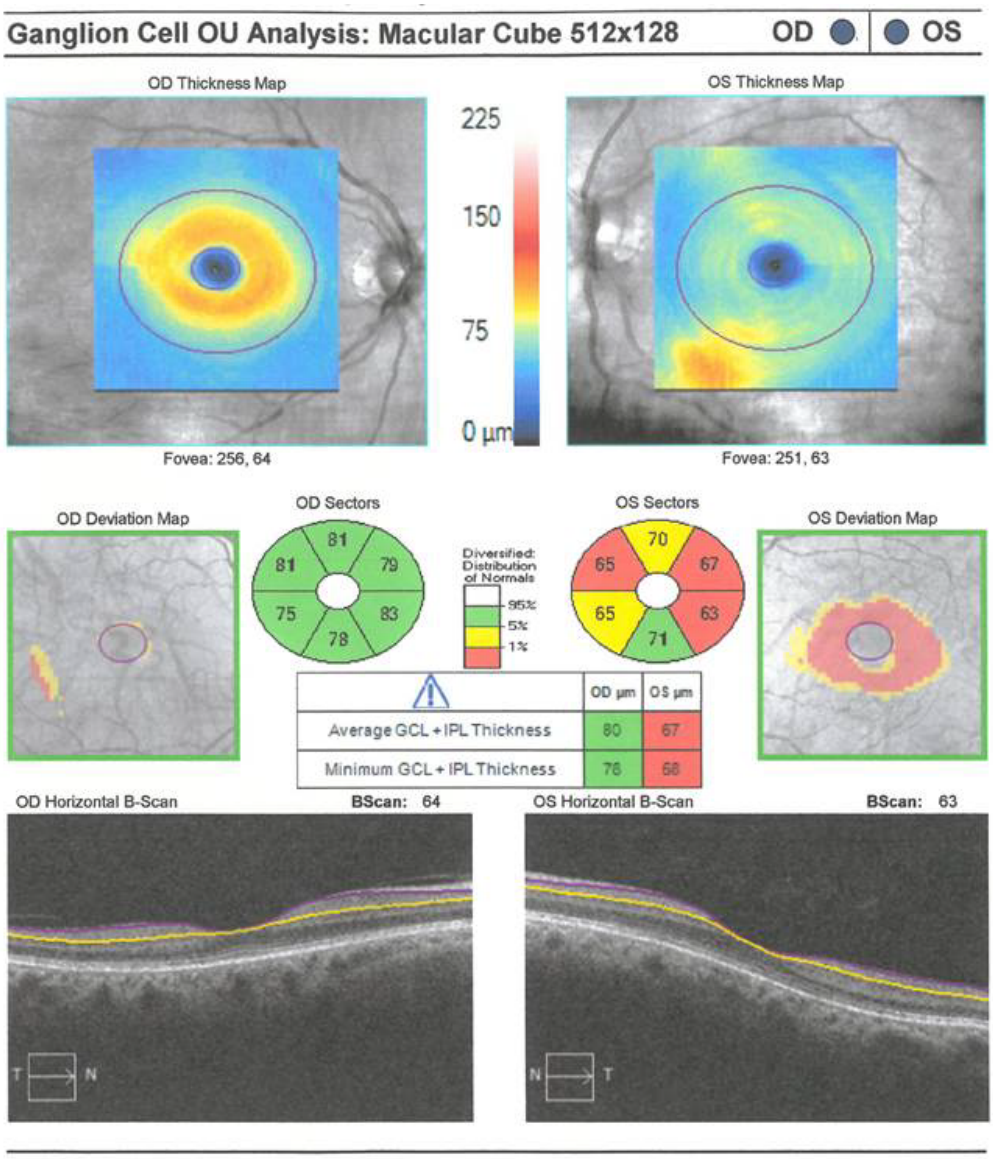

Figure 9: Follow up ganglion cell analysis showing thinning in the left eye.

capsular block, but IOP remained elevated for several days and then the NAION occurred, as was the case in this patient. In this case as well as those cases after cataract surgery, the prolonged IOP elevation induced ischemic optic neuropathy insult to the optic nerve was different than glaucomatous optic neuropathy as optic disc cupping did not occur.

YAG Capsulotomy is one of the most commonly performed ophthalmic laser procedures in the United States. The pre-operative and post-operative protocol varies widely among Ophthalmologists and may include topical IOP agent before and or after the procedure, topical steroid given immediately after procedure and for a few days or none at all. Follow up may include an IOP check at 30 to 60 min after the procedure and a return visit at one week to one month. It seems prudent then, that with the variability in protocols, a lower total YAG energy used per pulse and cumulatively during the capsulotomy may reduce the risk of postoperative IOP spike [7] cystoid macular edema, retinal detachments and NAION.

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