AN EYE CENTRAL CASE REPORT

A Case of Normal Tension Glaucoma

Managed by: Austin Ekeoba, O.D., Dipl. ABO
#215 – 12975 84 Ave, Queen Mary Park, Surrey V3W 1B3
www.eyecentral.ca

Abstract

Normal tension glaucoma (NTG) is a form of open-angle glaucoma characterized by progressive optic neuropathy and corresponding visual field loss despite intraocular pressures within the normal range (10 to 21 mmHg). Treatment involves deliberate IOP reduction through anti-glaucoma drop therapy, laser procedures such as ALT or SLT, or surgical management. This report explores the care process of a patient with normal tension glaucoma at Eye Central, covering diagnosis, treatment, management, and follow-up.

Keywords: Normal Tension Glaucoma, Intraocular Pressure, Optic Nerve, RNFL, Drance Hemorrhage

Introduction

Cranial nerve II, also known as the optic nerve, is a neural network that transmits visual impulses from the eye to the visual cortex and forms part of the visual pathway. Glaucoma is a disease of the optic nerve in which intraocular pressure is typically elevated, causing progressive damage to the nerve fibers and ultimately leading to vision loss if not diagnosed or properly managed. When glaucomatous optic neuropathy occurs in the presence of normal intraocular pressure, the condition is known as normal tension glaucoma (NTG).

Case Report

Initial Visit: December 5, 2024

A 44-year-old African American female presented to Eye Central for a routine eye examination with no visual or ocular concerns. Her last eye examination was conducted two years prior in New York, where she received an updated spectacle prescription for myopia. She had a history of contact lens wear but discontinued use due to dry eyes. There was no history of ocular surgery or trauma. She was using artificial tear lubricating drops as needed. Medical history was satisfactory. Family medical history was notable for diabetes and stroke (mother). Family ocular history was unknown. No known medication allergies or history of substance use were reported.

Uncorrected distance visual acuity was 20/150 OD and 20/100 OS. Uncorrected near acuity was 20/20 bilaterally. With habitual correction, visual acuity was 20/20- OD and 20/20 OS. Pupils were equal and responsive to light, with no afferent pupillary defect. Ocular motilities were full in both eyes, and confrontation visual field testing was full to finger count. Alternating cover testing revealed orthophoria at distance and 6 prism diopters of exophoria at near.

Objective refraction by autorefractor showed -1.75 -0.25 x 173 OD and -1.50 OS. Subjective refraction with a phoropter yielded -1.75 -0.25 x 180 OD and -1.50 OS, with a +1.25 ADD, achieving 20/20 at distance and near. Habitual glasses measured -1.50 OU by automated lensmeter.

Slit-lamp examination showed normal lids and lashes in both eyes. The cornea was clear with no superficial punctate keratitis. Tear break-up time with sodium fluorescein was 6 seconds bilaterally. The anterior chamber angle was open (Van Herick Grade III) with a deep, quiet anterior chamber. The iris appeared flat with no transillumination defects. Trace nuclear sclerosis of the crystalline lens was noted in both eyes. Intraocular pressure by Goldmann tonometry was 15 mmHg in both eyes.

The patient was dilated with 2.5% phenylephrine and 1.0% tropicamide at 10:11 am. Dilated fundus examination with a 90D lens revealed a large and suspicious optic nerve head (ONH) with a cup-to-disc ratio of 0.7 in both eyes. A Drance hemorrhage was noted at the inferior margin of the optic disc in the left eye (Fig. 1). The retina appeared flat and within normal limits bilaterally. The macula was healthy with a good foveal reflex. Binocular indirect ophthalmoscopy with a 20D lens showed no holes, tears, or hemorrhages in the peripheral retina.

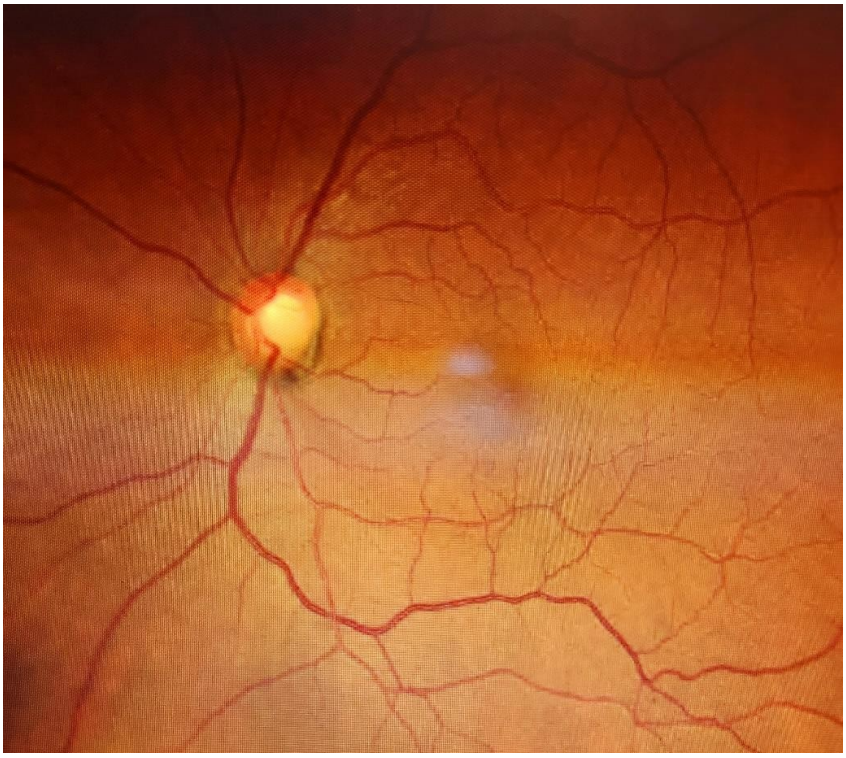

Fig. 1. Drance hemorrhage inferior OS.

Corneal pachymetry showed a central corneal thickness of 541 µm OD and 537 µm OS, with a corrective IOP value of +1 mmHg for both eyes, confirming the accuracy of the measured pressures.

Diagnosis and Management

A diagnosis of normal tension glaucoma (NTG) was established at this visit. Anti-glaucoma medication was initiated given the significant finding of a Drance hemorrhage in the left eye, indicating likely progression. Latanoprost 0.005% was prescribed at bedtime in both eyes. A target IOP of 10 to 12 mmHg was established, corresponding to approximately a 30% reduction from the baseline of 15 mmHg. The patient was scheduled to return for additional testing including gonioscopy, ONH OCT, ganglion cell complex (GCC) scan, and a 24-2 Humphrey visual field test.

Differential Diagnoses Considered

Primary Open-Angle Glaucoma (POAG): POAG presents similarly to NTG but with intraocular pressures exceeding 21 mmHg. Ruled out due to normal IOP measurements in both eyes using a Goldmann applanation tonometer. Pachymetry confirmed a normal central corneal thickness, reflecting accurate IOP estimation. Diurnal IOP patterns in POAG typically peak in the morning; this patient measured 15 mmHg at 10:11 am, within normal range.

Posterior Vitreous Detachment (PVD): A PVD can produce hemorrhage at the site of prior vitreoretinal attachment. Ruled out on dilated fundus examination and binocular indirect ophthalmoscopy, with no signs of vitreous detachment in either eye.

Ischemic Optic Neuropathy: Typically presents with disc pallor and possible disc hemorrhage, along with symptoms of pain, vision loss, or headache. The neuroretinal rim showed no pallor, and the patient presented with none of the associated systemic or ocular symptoms. Her age also made this diagnosis unlikely, as ischemic optic neuropathy most commonly affects individuals over 60.

Retinal Vascular Disease: Diabetic retinopathy, vein occlusion, and hypertensive retinopathy can all cause retinal hemorrhages. Dilated fundus examination and peripheral retinal assessment showed no hemorrhagic patterns consistent with retinal vascular disease, and the patient's systemic profile was unremarkable.

Second Visit: December 17, 2025

The patient returned for her scheduled follow-up. Corrected visual acuity was 20/20 in both eyes. IOP by Goldmann applanation tonometry was 10 mmHg OD and 11 mmHg OS, achieving the established target IOP range and confirming a treatment response of approximately 30% reduction from baseline.

ONH and RNFL analysis using OCT (Optic Disc Cube 200x200) showed normal RNFL in the right eye but an inferior RNFL dropout in the left eye, corresponding to the inferior Drance hemorrhage identified on fundoscopy (Fig. 2). The 24-2 Humphrey visual field report showed a non-specific field

defect OD and an arcuate field defect OS. Ganglion cell macular analysis (Macular Cube 512x128) revealed mild GCC thinning.

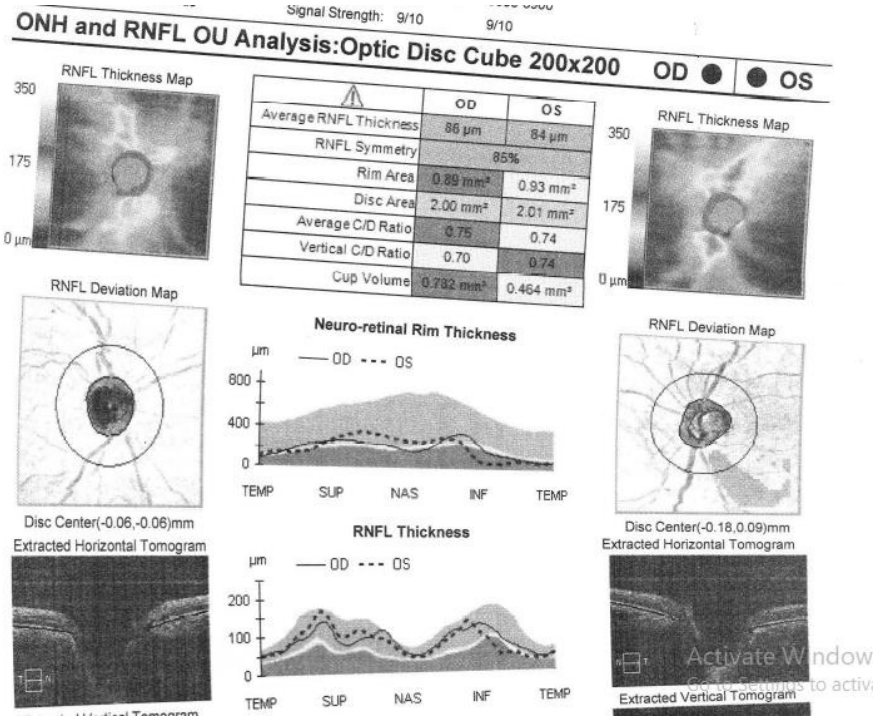

Fig. 2. ONH and RNFL OCT printouts showing inferior RNFL dropout OS.

Gonioscopy was performed using a Goldmann 3-mirror lens with 1.0% proparacaine and Genteal Gel as the coupling fluid. Anterior chamber angles were open in both eyes, with the most posterior visible structure corresponding to CBB/CBB/CBB/SP (S/N/I/T) OD and CBB/CBB/CBB/CBB (S/N/I/T) OS.

The patient was instructed to continue latanoprost 0.005% at bedtime in both eyes and to return in 3 months for an IOP check. Annual OCT of the ONH and GCC and a 24-2 visual field test were recommended to monitor for progression. Family members were encouraged to undergo glaucoma screening. A 3-month refill of latanoprost was sent to the patient's pharmacy at this visit.

Discussion

The intraocular pressure in patients with normal tension glaucoma is conventionally considered to range between 10 and 21 mmHg, despite the presence of glaucomatous nerve changes and corresponding visual field defects. There are also IOP-independent mechanisms that contribute to the pathophysiology of NTG, including vascular insufficiency at the optic nerve head, metabolic and neurodegenerative disorders, oxidative stress, and abnormal biomechanics of the lamina cribrosa. Genetic factors may also play a role; genes associated with NTG include optineurin (OPTN), TANK-binding kinase (TBK1), and myocilin (MYOC). Recognized risk factors include age, family history, female sex, nocturnal hypotension, systemic hypertension, migraine, and obstructive sleep apnea.

Research suggests a relationship between progressive optic neuropathy in NTG and underlying vascular insufficiency. Cardiovascular abnormalities implicated in NTG include hemodynamic crises, hypercoagulability, nocturnal systemic hypotension, elevated blood cholesterol and lipids, carotid artery disease, and slowed parapapillary, choroidal, and retinal circulation. Literature has also identified a link between immune-related conditions and NTG.

Diagnostic evaluation for NTG requires a careful and comprehensive ocular and systemic history. The history should document any previous episodes of elevated IOP, ocular trauma or inflammation, medication use (particularly steroids), sleep-related breathing disorders, and nocturnal blood pressure dips. Structural and functional differences from POAG include a thinner neuroretinal rim, particularly in the inferior and inferotemporal quadrant, more frequent disc hemorrhages, more prominent parapapillary atrophy in the beta zone, and visual field defects that tend to be closer to fixation and more focal in nature.

The Early Manifest Glaucoma Trial demonstrated that each mmHg reduction in IOP decreases glaucoma progression by 10%. The Collaborative Normal Tension Glaucoma Study showed that a 30% IOP reduction from baseline significantly slowed disease progression. This formed the basis for the target IOP set in this case. Methods for achieving IOP reduction include topical anti-glaucoma medications, selective laser trabeculoplasty (SLT), micro-invasive glaucoma surgery (MIGS), and augmented trabeculectomy in progressive cases. Prostaglandin analogues are typical first-line therapy. Brimonidine has comparable IOP-lowering efficacy to timolol with the added benefit of potential neuroprotective properties. In addition to IOP reduction, treating underlying cardiovascular risk factors to improve optic nerve head perfusion is an important component of comprehensive NTG management. Neuroprotective strategies including calcium channel blockers and ginkgo biloba extract are under investigation and show early promise.

Conclusion

Normal tension glaucoma presents unique diagnostic and management challenges given the absence of elevated intraocular pressure. Comprehensive diagnostic tools, including pachymetry, OCT, and visual field testing, are essential for detecting structural and functional optic nerve damage. Drance hemorrhage is a particularly significant prognostic indicator that should prompt further evaluation and consideration of more aggressive management. Treatment focuses on achieving a low target IOP while addressing systemic vascular risk factors. Regular monitoring through advanced imaging and perimetry is critical for detecting progression and adjusting the management plan to optimize long-term outcomes.

What This Means for You

Glaucoma is often called the "silent thief of sight" because it can cause irreversible vision loss before any symptoms are noticed. Normal tension glaucoma is a particularly subtle form, since it occurs even when eye pressure appears to be in the normal range. This makes routine comprehensive eye examinations especially important.

The good news is that when detected early, glaucoma can be managed effectively to preserve the vision you have. Prescription eye drops are often all that is needed to slow or stop progression. At Eye Central, we use advanced imaging and visual field testing to monitor your optic nerve with precision. If you have a family history of glaucoma, or if you have been told your optic nerve looks suspicious, we encourage you to book a comprehensive evaluation.

References

1. Heijl A, Leske MC, Bengtsson B, et al. Reduction of intraocular pressure and glaucoma progression: results from the Early Manifest Glaucoma Trial. Arch Ophthalmol. 2002;120(10):1268–79.

2. Mojon DS, Hess CW, Goldblum D, et al. High prevalence of glaucoma in patients with sleep apnea syndrome. Ophthalmology. 1999;106:1009–1012.

3. Adeghate J, Rahmatnejad K, Waisbourd M, Katz LJ. Intraocular pressure-independent management of normal tension glaucoma. Surv Ophthalmol. 2019;64(1):101–110.

4. Shields MB. Normal-tension glaucoma: Is it different from primary open-angle glaucoma? Curr Opin Ophthalmol. 2008;19:85–88.

5. Collaborative Normal-Tension Glaucoma Study Group. Comparison of glaucomatous progression between untreated patients with normal-tension glaucoma and patients with therapeutically reduced intraocular pressures. Am J Ophthalmol. 1998;126:487–497.

6. Gosling D, Meyer JJ. Normal Tension Glaucoma. StatPearls [Internet]. StatPearls Publishing; 2025.

7. Krupin T, Liebmann JM, Greenfield DS, et al. A randomized trial of brimonidine versus timolol in preserving visual function: results from the Low-Pressure Glaucoma Treatment Study. Am J Ophthalmol. 2011;151(4):671-681.

8. Killer HE, Pircher A. Normal tension glaucoma: review of current understanding and mechanisms of the pathogenesis. Eye (Lond). 2018;32(5):924-930.

9. Drance S, Anderson DR, Schulzer M. Risk factors for progression of visual field abnormalities in normal-tension glaucoma. Am J Ophthalmol. 2001;131(6):699-708.

10. Leske MC. Ocular perfusion pressure and glaucoma: clinical trial and epidemiologic findings. Curr Opin Ophthalmol. 2009;20(2):73-78.

11. Jonas JB, Aung T, Bourne RR, et al. Glaucoma. Lancet. 2017;390(10108):2183-2193.