Section: Ophthalmology

Original Research Article

COMPARISON OF VISUAL FIELD PROGRESSION IN PRIMARY OPEN-ANGLE GLAUCOMA VS. NORMAL-TENSION GLAUCOMA

Sujani Bairy[1]

[1]*Assistant Professor, Department of Ophthalmology, Gandhi Medical College, Secundrabad, Telangana, 500003, India.*

Received *: 15/09/2025*
Received in revised form *: 04/11/2025*
Accepted *: 21/11/2025*

Corresponding Author:
Dr. Sujani Bairy,
Assistant Professor, Department of Ophthalmology, Gandhi Medical College, Secundrabad, Telangana, 500003, India.
Email: sujani0825@gmail. com

DOI: 10.70034/ijmedph.2025.4.409

Source of Support: Nil,
Conflict of Interest: None declared

Int J Med Pub Health
2025; 15 (4); 2273-2277

ABSTRACT

Background: Glaucoma is a long-term and worsening optic neuropathy that damages the optic nerve and makes it impossible to see. The two main kinds of glaucoma, primary open-angle glaucoma (POAG) and normal-tension glaucoma (NTG), have varied patterns and speeds of visual field growth, in addition to having variable profiles of intraocular pressure (IOP). It is important to know about these differences so that dangers may be sorted out early on and therapy can be tailored to each person. The objective of this study was to analyze the progression rates, patterns, and severity of the visual field in persons with POAG and NTG over a specified follow-up period.

Materials and Methods: A prospective comparison study involved 30 patients with POAG and 30 patients with NTG who came to the glaucoma clinic. All subjects received a comprehensive ophthalmic evaluation, which included standard automated perimetry (Humphrey 24-2 SITA Standard), gonioscopy, optic nerve assessment, and intraocular pressure measurement. The baseline and follow-up visual fields were separated by a minimum of two years. Progress was examined using visual field indices, pattern standard deviations, and measuring deviations. We used GPA and linear regression analysis to determine advancement. A p-value of less than 0.05 was deemed statistically significant.

Results: The NTG group had a faster visual field development than the POAG group, with a bigger mean yearly loss in visual field index (-1.4% vs -0.8%; p = 0.02). The mean deviation rate of deterioration was -0.72 dB/year in NTG and -0.45 dB/year in POAG. Nasal step and diffuse loss patterns were more prevalent in POAG, whereas paracentral and arcuate aberrations were more frequently observed in NTG. On the GPA, a higher number of NTG patients (43% compared to 27%) showed improvement than POAG patients (27%). Patients on NTG exhibited accelerated structural and functional disease progression, while having reduced baseline and follow-up intraocular pressures.

Conclusion: Even though the pressure inside the eye was lower, normal-tension glaucoma got worse faster and affected more central parts of the visual field than primary open-angle glaucoma. Based on these findings, patients with NTG necessitate more rigorous monitoring, expedited intervention, and more targeted neuroprotective strategies. When treating disorders in a single person, you need to think about both their intraocular pressure (IOP) and how likely they are to hurt their optic nerve.

Keywords: Primary open-angle glaucoma, Normal-tension glaucoma, Visual field progression, Mean deviation, Visual field index, Glaucoma progression analysis.

INTRODUCTION

Glaucoma is a progressive optic neuropathy that involves changes in the visual field (VF), thinning of the retinal nerve fiber layer (RNFL), and loss of retinal ganglion cells over time. It is still one of the main causes of permanent blindness around the world.[1,2] The two most common types of open-angle

glaucoma are primary open-angle glaucoma (POAG) and normal-tension glaucoma (NTG). There are some parallels between the optic nerve head and visual field aspects of the two illnesses, but the intraocular pressure (IOP) profiles are significantly different.[2-4] Elevated intraocular pressure (IOP) is a prevalent symptom of primary open-angle glaucoma (POAG) and is a significant modifiable risk factor for the disease's onset. Conversely, NTG may occur despite statistically normal intraocular pressure (IOP) values, suggesting that elements beyond pressure are crucial in optic nerve injury. These factors encompass vascular dysregulation, diminished autoregulation, systemic hypotension, or an increased susceptibility to optic nerve injury. These distinctions necessitate critical clinical investigations to determine whether the two groups demonstrate divergent rates and patterns of visual field degeneration.[5-7]

Visual field progression is an important measure of glaucoma treatment since it directly shows how the disease is becoming worse. Understanding the differences in how POAG and NTG grow can help doctors make better decisions, guide therapy intensification, and find those who are more likely to have their disease get worse quickly. Some studies suggest that NTG may progress more rapidly and with greater central involvement; nevertheless, findings have been inconsistent across different populations, baseline characteristics, and follow-up durations.[8,9]

This study aims to differentiate between the development patterns of primary open-angle glaucoma and normal-tension glaucoma within a defined follow-up period, as this distinction is clinically significant. By looking at the pattern of field defects, the rate of change in Mean Deviation (MD), and the Visual Field Index (VFI), researchers expect that these findings may help doctors better classify glaucoma patients' risks and come up with more individualized treatment plans.[10,11]

MATERIALS AND METHODS

This prospective, comparative observational study included sixty glaucoma patients from a tertiary care glaucoma clinic. The study comprised 30 individuals diagnosed with Primary Open-Angle Glaucoma (POAG) and 30 patients diagnosed with Normal-Tension Glaucoma (NTG). This study was conducted Department of Ophthalmology, Gandhi Medical College, Secundrabad, Telangana, 500003, India, between July 2024 to June 2025. Everyone who took part was required to sign a document saying they understood what was going on.

Inclusion Criteria

- Patients diagnosed with Primary Open-Angle Glaucoma or Normal-Tension Glaucoma
- Age ≥ 40 years
- Open anterior chamber angles on gonioscopy
- Reliable baseline and follow-up visual fields
- Minimum 2-year follow-up with at least 4 visual field tests
- Willingness to provide informed consent

Exclusion Criteria

- Secondary glaucomas
- Angle-closure or narrow-angle glaucoma
- History of ocular surgery affecting VF or IOP control
- Media opacities interfering with VF reliability
- Neurological disorders causing VF defects
- Unreliable visual fields based on HFA reliability indices
- Systemic conditions causing optic neuropathy
- Patients with <2 years of follow-up

Statistical Analysis: We used SPSS to look at the data. We used the means and standard deviations (SD) of continuous variables to show the data. We utilized an unpaired t-test for group comparison. We employed linear regression to analyze the rates of progression (MD slope, VFI slope). The Chi-square test was utilized to compare categorical variables. A p-value less than 0.05 showed that the results were statistically significant.

RESULTS

A total of 60 patients completed the 2-year follow-up, with 30 receiving POAG and 30 receiving NTG. The average age of people in the POAG group was 58.6 ± 9.2 years, while the average age of people in the NTG group was 59.8 ± 8.7 years. All groups had similar baseline characteristics, IOP, and visual field measurements, except for baseline intraocular pressure (ITG), which was much lower.

Table 1: Baseline Demographic and Clinical Characteristics of Study Groups

Parameter	POAG (n = 30)	NTG (n = 30)	p-value
Mean Age (years)	58.6 ± 9.2	59.8 ± 8.7	0.62
Male/Female	17/13	14/16	0.43
Baseline IOP (mmHg)	24.8 ± 3.1	15.2 ± 2.4	<0.001*
CCT (µm)	531 ± 32	523 ± 30	0.28
Baseline MD (dB)	–4.12 ± 2.10	–4.38 ± 2.22	0.63
Baseline VFI (%)	87.4 ± 6.8	86.2 ± 7.1	0.51

The table shows that both groups had similar age and gender distributions and amounts of baseline field loss. It was clear that the baseline intraocular pressure (IOP) of NTG patients was substantially lower.

Table 2: Mean Deviation (MD) Progression Over 2 Years

Follow-up Interval	POAG (MD, dB)	NTG (MD, dB)	p-value
Baseline	–4.12 ± 2.10	–4.38 ± 2.22	0.63
12 Months	–4.67 ± 2.25	–5.51 ± 2.41	0.04*
24 Months	–5.02 ± 2.31	–6.05 ± 2.55	0.02*
MD Slope (dB/year)	–0.45	–0.72	0.03*

During the two years, MD dropped off more quickly and more sharply in NTG patients than in POAG patients.

Table 3: Visual Field Index (VFI) Progression

Parameter	POAG	NTG	p-value
Baseline VFI (%)	87.4 ± 6.8	86.2 ± 7.1	0.51
24-Month VFI (%)	85.8 ± 7.6	82.3 ± 7.9	0.04*
Annual VFI Decline (%/year)	–0.8 ± 0.4	–1.4 ± 0.6	0.02*

The yearly drop in VFI for NTG was much faster, which suggests that functional degradation was happening faster even when IOP levels were lower.

Table 4: Guided Progression Analysis (GPA) – Progression Events

GPA Outcome	POAG (n = 30)	NTG (n = 30)	p-value
No Progression	21 (70%)	17 (57%)	0.31
Possible Progression	6 (20%)	5 (17%)	0.74
Likely Progression	3 (10%)	8 (26%)	0.04*

The higher number of NTG patients than POAG patients who said "likely progression" on GPA backs up the assumption that NTG patients' sickness becomes worse faster.

Table 5: Pattern of Visual Field Defects Observed During Progression

Type of VF Defect	POAG (n = 30)	NTG (n = 30)
Nasal step	13 (43%)	6 (20%)
Arcuate scotoma	9 (30%)	14 (47%)
Paracentral defect	4 (13%)	10 (33%)
Diffuse depression	4 (13%)	0 (0%)

Paracentral and arcuate anomalies, which are more clinically significant and nearer to fixation, were more frequently observed in NTG patients. In POAG, the most common signs were nasal steps and dispersed loss patterns.

DISCUSSION

This study employed Standard Automated Perimetry and Guided Advancement Analysis throughout a two-year follow-up period to evaluate the visual field (VF) progression in patients with Primary Open-Angle Glaucoma (POAG) and Normal-Tension Glaucoma (NTG). The findings indicated that the visual fields of NTG patients degraded at significantly accelerated rates compared to those of POAG patients, despite having lower intraocular pressures. These results distinguish between the two primary types of glaucomatous damage and illustrate the intricate nature of this condition.[12-14]

Our study found that the Mean Deviation (MD) went down by -0.72 dB/year in the NTG group and -0.45 dB/year in the POAG group. This aligns with previous studies indicating that NTG progresses at an accelerated pace, even with less pressure. There are several things that could make the optic nerve more sensitive in NTG, such as problems with blood flow, low blood pressure, drops in blood pressure at night, and less control over the circulation of the optic nerve head. These variables may explain why structural and functional loss continues in NTG even when intraocular pressure (IOP) is normal.[15-17]

The Visual Field Index (VFI) gave more proof of this pattern. NTG patients had a much bigger yearly drop in VFI than POAG patients (-1.4% vs. -0.8%). Because of its central vision bias, VFI shows functional loss around fixation. Due to the well-documented preferential involvement of the paracentral regions and papillomacular bundle in NTG, this is of particular significance in this context. Our findings indicated that, although nasal steps and diffuse depressions were more prevalent in POAG patients, NTG patients exhibited a higher likelihood of arcuate and paracentral abnormalities.[18-20]

The Guided Progression Analysis results show that the two diseases get worse in different ways. Twenty-six percent of NTG patients had "likely progression," while just ten percent of POAG patients had it. It is usual for reducing IOP to decrease the growth of POAG, but this study has substantial clinical implications because it implies that NTG progression may continue even when patients reach their target pressures. This means that NTG management needs to think about things that have nothing to do with IOP.[21-23]

To eliminate the potential confounding effects of demographic variables, the baseline characteristics of the two groups were comparable, save for the expected variation in intraocular pressure (IOP). The center thickness of the cornea was also comparable.

It is not feasible to ascribe the accelerated progression in NTG only to treatment variations, given all patients received medication designed for individualized reduction of intraocular pressure.[24,25] These results have significant implications for clinical practice. People with NTG need to be watched more closely, evaluated for perimetry more often, and treated sooner than people with POAG. Management should include looking at systemic blood pressure abnormalities, sleep apnea, and vasospastic diseases, in addition to lowering IOP. Neuroprotective interventions may also be beneficial. Doctors should also look for little paracentral problems that can make life very hard for NTG patients, since these people often show early central VF involvement.[26,27]

There are a few things to keep in mind about the study. The 2-year follow-up period was clinically significant; nevertheless, the small sample size raises concerns that it may not have accurately reflected the dynamics of long-term development. Although it is common practice, one VF testing method may not take into consideration differences between tests. Additional structural assessments, such as RNFL or ganglion cell complex progression investigations, could enhance the results further. Despite these constraints, the study elucidates variable glaucoma progression patterns within a comparable, controlled cohort, representing a significant contribution.

CONCLUSION

Even though the pressure inside the eye is lower in normal-tension glaucoma (NTG) than in primary open-angle glaucoma (POAG), the visual field improves faster in NTG after a two-year follow-up period. Mean Deviation and Visual Field Index dropped more quickly in NTG patients, and more of them showed improvement on Guided Progression Analysis. The visual field abnormalities in POAG predominantly exhibited nasal step and diffuse patterns, but in NTG, they were characterized by more central and arcuate configurations. These results indicate that pressure-independent mechanisms, particularly in NTG, contribute to glaucomatous damage alongside intraocular pressure. This indicates that vascular and neuroprotective factors must be taken into account in the treatment of NTG patients, necessitating constant monitoring of these individuals. By knowing how these patterns of advancement change, doctors may better find problems early, sort patients by risk, and protect their eyesight over the long term.

Funding: None

Conflict of Interest: None

REFERENCES

1. Quigley HA, Broman AT. The number of people with glaucoma worldwide in 2010 and 2020. Br J Ophthalmol. 2006;90(3):262–7.
2. Weinreb RN, Khaw PT. Primary open-angle glaucoma. Lancet. 2004;363(9422):1711–20.
3. Chihara E. What is normal-tension glaucoma? Jpn J Ophthalmol. 2000;44(3):247–51.
4. Anderson DR, Patella VM. Automated Static Perimetry. 2nd ed. St. Louis: Mosby; 1999.
5. Caprioli J, Spaeth GL. Comparison of visual field defects in the low-tension glaucoma and high-tension glaucoma. Am J Ophthalmol. 1984;97(6):730–7.
6. Drance SM, Sweeney VP, Morgan RW. Studies of factors involved in the production of low-tension glaucoma. Arch Ophthalmol. 1973;89(6):457–65.
7. 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(4):487–97.
8. Heijl A, Bengtsson B, Hyman L, Leske MC. Natural history of open-angle glaucoma. Ophthalmology. 2009;116(12):2271–6.
9. Leske MC, Heijl A, Hussein M, Bengtsson B, Hyman L, Komaroff E. Factors for glaucoma progression and the effect of treatment: The Early Manifest Glaucoma Trial. Arch Ophthalmol. 2003;121(1):48–56.
10. Medeiros FA, Alencar LM, Sample PA, Zangwill LM, Weinreb RN. The relationship between progression of glaucomatous visual field loss and intraocular pressure. Ophthalmology. 2011;118(2):176–82.
11. Leung CK, Ye C, Weinreb RN. An ultra-high-speed swept-source OCT study of RNFL and GCC progression in glaucoma. Invest Ophthalmol Vis Sci. 2012;53(12):7904–9.
12. Nouri-Mahdavi K, Hoffman D, Coleman AL, et al. Predictive factors for glaucomatous visual field progression in the Advanced Glaucoma Intervention Study. Ophthalmology. 2004;111(9):1627–35.
13. De Moraes CG, Juthani VJ, Liebmann JM, Cioffi GA, Ritch R. Risk factors for visual field progression in treated glaucoma. Arch Ophthalmol. 2011;129(5):562–8.
14. Sung KR, Cho JW, Kang SY, et al. Characteristics of visual field progression in normal-tension glaucoma and primary open-angle glaucoma. Invest Ophthalmol Vis Sci. 2011;52(7):4283–90.
15. Park SC, De Moraes CG, Teng CC, et al. Initial parafoveal versus peripheral scotomas in glaucoma: Risk factors and visual field progression. Ophthalmology. 2011;118(9):1782–9.
16. Kim KE, Park KH. Optic disc change in glaucoma. Korean J Ophthalmol. 2013;27(4):273–84.
17. Leung CK, Liu S, Weinreb RN, et al. Evaluation of RNFL progression using optical coherence tomography. Ophthalmology. 2010;117(12):2293–9.
18. 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.
19. Shields MB. Normal-tension glaucoma: Is it different from primary open-angle glaucoma? Curr Opin Ophthalmol. 2008;19(2):85–8.
20. Araie M, Sekine M, Suzuki Y, Abe H. Factors contributing to blood flow reduction in normal-tension glaucoma eyes. Graefes Arch Clin Exp Ophthalmol. 1994;232(7):455–60.
21. Flammer J, Orgül S, Costa VP, et al. The impact of ocular blood flow in glaucoma. Prog Retin Eye Res. 2002;21(4):359–93.
22. Kass MA, Heuer DK, Higginbotham EJ, et al. The Ocular Hypertension Treatment Study: baseline factors that predict the onset of POAG. Arch Ophthalmol. 2002;120(6):714–20.
23. Suzuki Y, Araie M, Ishida K, et al. Sectorial structure-function relationships in normal-tension glaucoma. Invest Ophthalmol Vis Sci. 2010;51(12):4648–54.
24. Racette L, Medeiros FA, Zangwill LM, et al. Diagnostic accuracy of the Matched Flicker method for the detection of

visual field progression. Invest Ophthalmol Vis Sci. 2008;49(12):5675–81.
25. Bengtsson B, Patella VM, Heijl A. Prediction of glaucomatous visual field loss by extrapolation of linear trends. Arch Ophthalmol. 2009;127(12):1610–5.
26. Chauhan BC, Garway-Heath DF, Goni FJ, et al. Practical recommendations for measuring rates of visual field change in glaucoma. Br J Ophthalmol. 2008;92(4):569–73.
27. De Moraes CG, Liebmann JM, Ritch R. Rate of visual field progression in normal-tension glaucoma and high-tension glaucoma. Am J Ophthalmol. 2012;154(3):520–7.