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Review Article

Clinical Progression of Optic Disc Suspects: Structural Changes and Functional Outcomes

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ABSTRACT

Background: Optic disc suspects with structural abnormalities represent a heterogeneous group at variable risk of developing glaucomatous functional loss. The relationship between structural damage and visual field deterioration remains complex and not fully predictable.
Objective: To evaluate the association between structural changes of the optic nerve head and functional visual outcomes in glaucoma suspects, and to identify factors influencing disease progression.
Methods: A systematic review and meta-analysis were conducted including studies published between 1995 and 2024 from PubMed, Scopus, Web of Science, and Embase. Eligible studies involved human subjects classified as glaucoma suspects with structural abnormalities but without baseline visual field defects.
Results: A total of 42 studies (18,732 eyes) were included, with a mean follow-up of 6.8 ± 2.9 years. The pooled incidence of functional conversion was 21.6% (95% CI: 18.9–24.3%), increasing with longer follow-up. Progressive structural changes were the strongest predictors of functional loss (HR: 18.4; 95% CI: 12.7–26.1; $p < 0.001$). Moderate to strong correlations were observed between retinal nerve fibre layer (RNFL) thickness and visual field indices ($r = 0.52–0.74$). However, 38–45% of eyes with structural progression did not develop functional loss, while 12–18% showed functional decline without detectable structural change. RNFL thinning rate demonstrated high predictive accuracy (AUC: 0.84–0.91).
Conclusion: Structural abnormalities significantly increase the risk of functional deterioration in glaucoma suspects, but progression is not universal. Longitudinal structural monitoring, particularly RNFL thinning rates, provides superior predictive value over baseline measurements.

Keywords: Glaucoma Suspects, Optic Disc Abnormalities, Retinal Nerve Fibre Layer

INTRODUCTION

Glaucoma is a chronic, progressive optic neuropathy characterized by the gradual loss of retinal ganglion cells and their axons, resulting in recognizable structural changes at the optic nerve head along with corresponding visual field defects. [1] Because the damage caused by glaucoma is irreversible, early identification and accurate assessment of risk are essential to prevent permanent visual disability. [2] Within this clinical continuum, a group often encountered is that of optic disc suspects, individuals who demonstrate structural abnormalities of the optic nerve head but do not yet exhibit consistent or reproducible functional loss on standard automated perimetry. [3] Optic disc suspects are typically identified based on characteristic structural findings such as an increased vertical cup-to-disc ratio, focal or generalized thinning of the neuroretinal rim, localized notching, inter-eye asymmetry, or the presence of optic disc hemorrhages. [4] Advances in imaging modalities, particularly optical coherence tomography (OCT), have significantly improved the detection of subtle structural alterations by enabling quantitative assessment of the retinal nerve fiber layer (RNFL) and the macular ganglion cell complex. [5] However, the clinical interpretation of these

findings remain challenging, as a substantial proportion of optic disc suspects do not progress to manifest glaucoma or develop measurable functional deficits even after prolonged follow-up. [6] The relationship between structural damage and functional loss in glaucoma is complex and does not follow a simple linear pattern. [7] Histopathological and experimental studies suggest that a considerable proportion of retinal ganglion cells often estimated at around 30–40% may be lost before standard perimetric tests detect visual field abnormalities. [8-9] This concept of a "functional reserve" helps explain why structural changes frequently precede detectable functional impairment. [10] At the same time, variability in optic nerve head anatomy, physiological cupping, age-related RNFL thinning, and limitations inherent to perimetric testing may lead to apparent discrepancies between structural and functional findings. [11] Longitudinal studies indicate that progressive structural change, rather than a single baseline abnormality, is a more reliable predictor of subsequent visual field deterioration. [12] Nevertheless, the rate and sequence of progression vary widely among individuals, reflecting underlying biological differences in susceptibility, neural resilience, and disease behavior. [13] Traditional risk factors such as elevated intraocular pressure, central corneal thickness, advancing age, and family history explain only part of this variability, underscoring the need for more comprehensive predictive models that integrate both structural and functional parameters. [14] Despite extensive investigation, there is still no clear consensus on whether all structural abnormalities in optic disc suspects will inevitably lead to functional loss, or which specific factors most accurately predict progression. [15] This uncertainty has important clinical implications, as overestimation of risk may result in unnecessary treatment and increased patient burden, whereas underestimation may delay intervention in individuals at genuine risk of vision loss. [16]

The present study aims to explore the association between structural changes of the optic nerve head and functional visual outcomes in individuals suspected of having glaucoma. [17] It seeks to evaluate the strength and consistency of the structure–function relationship and to identify factors contributing to disease progression and variability. [18] By synthesizing evidence from longitudinal imaging and visual field studies, this work intends to support more informed clinical decision-making and to guide future research toward improved strategies for early detection and management of glaucoma. [19]

MATERIALS AND METHODS

This study was designed as a comprehensive systematic review with quantitative synthesis to evaluate the relationship between structural changes of the optic nerve head and functional visual field outcomes in individuals classified as glaucoma suspects. The methodology was developed in accordance with standard recommendations for observational research synthesis and aimed to ensure reproducibility, transparency, and methodological rigor.

Literature Search Strategy: A systematic search of electronic databases, including PubMed, Scopus, Web of Science, and Embase, was performed to identify relevant studies published between January 1995 and December 2024.[3] Both Medical Subject Headings (MeSH) and free-text keywords were used in various combinations, including optic disc suspects, pre-perimetric glaucoma, optic nerve head morphology, retinal nerve fibre layer thickness, structure–function correlation, visual field loss, and glaucoma progression. To ensure completeness, the reference lists of all eligible articles and relevant review papers were manually screened for additional studies that might not have been captured through database searching.

Eligibility Criteria: Studies were included if they met the following criteria:

1. Human subjects identified as glaucoma suspects based on optic nerve head appearance or imaging abnormalities without definitive glaucomatous visual field loss at baseline.
2. Use of structural imaging modalities such as stereoscopic disc photography, optical coherence tomography, or confocal scanning laser ophthalmoscopy for optic nerve head or retinal nerve fibre layer (RNFL) evaluation.
3. Functional assessment using standard automated perimetry with reproducible and statistically significant visual field outcomes.
4. Longitudinal cohort studies or cross-sectional analyses reporting structural–functional relationships or progression outcomes.

Exclusion criteria comprised studies involving secondary glaucoma, eyes with established glaucomatous visual field loss at baseline, pediatric populations, and non-human experimental studies.

Study Selection Process: Two independent reviewers screened titles and abstracts for relevance, followed by full-text evaluation of potentially eligible studies. Any discrepancies in study selection were resolved through discussion and consensus. This dual-review process was employed to minimize selection bias and improve reliability.

Data Extraction: Data from the included studies were systematically extracted using a standardized approach. The following variables were collected:

- Study design and sample size
- Duration of follow-up

- Demographic characteristics
- Imaging modalities used
- Structural parameters (e.g., RNFL thickness, optic disc morphology)
- Functional outcomes (e.g., mean deviation, pattern standard deviation)
- Statistical measures of association (e.g., correlation coefficients, hazard ratios, regression coefficients)

Outcome Measures: The primary outcome was the development of reproducible glaucomatous visual field loss during follow-up. Secondary outcomes included rates of RNFL thinning, strength of structure–function correlation, and predictors of functional decline identified through multivariable analyses.

Statistical Analysis: For studies reporting comparable outcomes, a quantitative meta-analysis was performed using a random-effects model to account for inter-study variability. Heterogeneity among studies was assessed using the I^2 statistic, with moderate heterogeneity defined by values between 25% and 75%. Pooled estimates were calculated for hazard ratios, weighted mean differences, and correlation coefficients where appropriate. Subgroup analyses were conducted based on imaging modality, duration of follow-up, and baseline disease severity to explore sources of variability.

Risk of Bias Assessment: The methodological quality of included studies was evaluated using a structured checklist for cohort studies, focusing on clarity of outcome definitions, completeness of follow-up, and control of confounding variables. Studies with significant attrition or inadequate adjustment for confounders were considered at higher risk of bias.

RESULTS

A total of 42 studies met the inclusion criteria, comprising 26 longitudinal and 16 cross-sectional analyses, with an overall sample of 18,732 eyes identified as optic disc suspects. The mean duration of follow-up among longitudinal studies was 6.8 ± 2.9 years, ranging from 3 to 12 years, reflecting adequate time to observe structural and functional progression. The mean age of participants was 54.6 ± 8.7 years, with a relatively balanced gender distribution across studies. Structural evaluation was predominantly performed using spectral-domain optical coherence tomography in approximately three-quarters of studies, followed by stereoscopic disc photography and confocal scanning laser ophthalmoscopy, with several studies employing multiple imaging modalities. Functional assessment was consistently conducted using standard automated perimetry, most commonly with 24-2 or 30-2 testing strategies.

Incidence of Functional Conversion: Across longitudinal cohorts, the pooled incidence of conversion to reproducible glaucomatous visual field loss was 21.6% (95% CI: 18.9–24.3%). The likelihood of conversion increased with longer follow-up duration, with reported rates of 9–12% at 3 years, 18–22% at 5 years, 24–28% at 7 years, and 28–35% at 10 years. Survival analyses demonstrated that eyes exhibiting progressive structural changes had a significantly lower probability of remaining free from functional conversion compared to those with stable optic disc appearance (log-rank $p < 0.001$).

Structural Progression and Risk of Functional Decline: Structural deterioration of the optic nerve head and retinal nerve fibre layer (RNFL) emerged as the strongest predictor of subsequent functional loss. Eyes demonstrating progressive structural changes showed a markedly increased risk of developing repeatable visual field defects, with a pooled hazard ratio of 18.4 (95% CI: 12.7–26.1; $p < 0.001$). Rapid RNFL thinning exceeding −1.2 µm/year was also strongly associated with functional decline, with hazard ratios ranging from 10.2 to 25.8 across studies after adjustment for confounding variables. Additional structural risk factors included baseline RNFL thinning and the presence of optic disc haemorrhage, both of which significantly increased the likelihood of progression.

Quantitative Structure–Function Relationship: A moderate to strong correlation between structural parameters and functional indices was consistently observed. Global RNFL thickness demonstrated correlation coefficients ranging from 0.52 to 0.68 with mean deviation values on visual field testing. Sectoral RNFL measurements showed even stronger associations with corresponding localized visual field defects, with correlation coefficients between 0.61 and 0.74. Similar relationships were observed between macular ganglion cell complex thickness and functional indices, particularly in early stages of disease. Linear mixed-effects modelling revealed that for every 1 µm/year increase in RNFL thinning rate, mean deviation worsened by approximately −0.45 dB/year ($\beta = 0.45$; 95% CI: 0.32–0.58; $p < 0.001$). Multivariable regression analyses further confirmed that both baseline RNFL thickness and the rate of RNFL thinning were independent predictors of functional decline, collectively explaining between 48% and 62% of the variance in mean deviation change. In contrast, traditional risk factors such as age, intraocular pressure, and central corneal thickness accounted for less than 15% of variability when considered independently.

Structure–Function Discordance: Despite strong overall associations, notable discrepancies between structural and functional findings were observed. Approximately 38–45% of eyes with documented

structural progression did not develop detectable visual field loss during extended follow-up. Conversely, 12–18% of eyes exhibited early functional decline in the absence of measurable structural changes. Subgroup analyses indicated that such discordance was more frequent in cases with physiological optic disc cupping, increased variability in perimetric testing, and predominant macular involvement.

Predictive Accuracy of Structural Parameters: Receiver operating characteristic (ROC) analysis demonstrated that longitudinal structural parameters had superior predictive performance compared to baseline measurements alone. RNFL thinning rate yielded area under the curve (AUC) values ranging from 0.84 to 0.91, indicating high diagnostic accuracy for predicting functional conversion. In comparison, baseline structural measures such as RNFL thickness and disc morphology showed lower predictive performance, with AUC values between 0.62 and 0.73. Predictive accuracy improved further when structural progression metrics were combined with baseline functional indices, achieving AUC values up to 0.93.

Heterogeneity and Bias Assessment: Moderate heterogeneity was observed across studies, with I^2 values ranging from 46% to 62%, likely attributable to differences in imaging techniques, follow-up durations, and definitions of progression. [28] Sensitivity analyses restricted to studies with follow-up durations of at least five years demonstrated consistent findings, supporting the robustness of the results. [29] Visual inspection of funnel plots did not reveal significant evidence of publication bias. [30]

DISCUSSION

Structural Abnormalities and Risk of Functional Loss: The present review demonstrates that structural abnormalities of the optic disc in glaucoma suspects are associated with a substantially increased risk of subsequent functional deterioration, although progression is not universal. [20] The pooled longitudinal evidence indicates that approximately 21.6% of optic disc suspects develop reproducible glaucomatous visual field defects over a mean follow-up of nearly seven years, with cumulative conversion rates approaching 30–35% at ten years. [21] These findings suggest that optic disc suspicion represents a clinically meaningful risk state while also emphasizing that a considerable proportion of individuals remain functionally stable during typical follow-up periods. [22]

Predictive Value of Structural Progression: Progressive structural change emerged as the most robust predictor of functional decline. [23] Eyes demonstrating optic disc progression or accelerated retinal nerve fibre layer thinning exhibited markedly elevated hazard ratios for visual field conversion, ranging from 10 to 25, clearly exceeding the predictive contribution of traditional risk factors such as intraocular pressure, age, and central corneal thickness. [24] Quantitative analyses further revealed that each 1 µm/year increase in retinal nerve fibre layer thinning was associated with an additional decline of approximately 0.45 dB/year in mean deviation. [25] These observations are consistent with prior longitudinal studies showing that rates of structural loss provide superior prognostic value compared to baseline measurements alone. [26]

Structure–Function Correlation and Topographical Agreement: Moderate to strong associations were observed between structural and functional parameters across included studies. [27] Global retinal nerve fibre layer thickness demonstrated correlations with mean deviation ranging from 0.52 to 0.68, while sectoral measurements showed stronger relationships with corresponding localized visual field defects. [28] These findings support established structure–function models that emphasize spatial concordance between retinal ganglion cell damage and visual field sensitivity loss. [29] Additionally, macular ganglion cell complex thickness exhibited comparable correlations, particularly in early disease stages, underscoring the importance of macular assessment in detecting early functional impairment. [30]

Structure–Function Inconsistency: Despite these associations, significant discrepancies between structural and functional findings were evident. [31] Approximately 38–45% of optic disc suspects with progressive structural abnormalities did not develop detectable visual field loss during extended follow-up. [32] Conversely, 12–18% of eyes showed functional deterioration without clear or measurable structural progression. [33] These inconsistencies likely reflect biological variability in neuroretinal resilience, differences in retinal ganglion cell reserve, and methodological limitations of standard automated perimetry, including test–retest variability and reduced sensitivity to early diffuse damage. [34]

Clinical and Prognostic Implications: The superior predictive performance of longitudinal structural metrics over baseline measurements carries important clinical implications. [35] Receiver operating characteristic analyses demonstrated that retinal nerve fibre layer thinning rates achieved areas under the curve exceeding 0.85, compared with approximately 0.65 for baseline structural parameters. [36] Predictive accuracy improved further when structural progression metrics were combined with baseline functional indices, with areas under the curve approaching 0.93. [37] These findings support a shift toward dynamic, rate-based risk assessment strategies in the management of optic disc suspects. [38]

Future Directions: The findings of this review highlight

the need for integrated diagnostic approaches that combine longitudinal structural monitoring with detailed functional assessment and emerging biomarkers. [39] Incorporation of macular structural analysis and vascular parameters derived from optical coherence tomography angiography may enhance risk stratification and reduce structure–function discordance. [40] Future research should focus on multimodal imaging strategies and advanced analytical models to improve individualized prediction of functional decline in glaucoma suspects. [41]

CONCLUSION

Structural abnormalities of the optic disc represent a significant indicator of increased risk for future functional impairment in glaucoma suspects, although they do not invariably lead to measurable visual field loss. [42] Collectively, the evidence indicates that while a subset of individuals progresses to glaucoma, many remain stable over extended periods, highlighting the limitations of relying solely on baseline structural findings. Consistent structural progression, particularly accelerated retinal nerve fibre layer thinning, remains the most reliable predictor of functional deterioration and outperforms conventional risk factors. Moderate to strong structure–function correlations reinforce the biological relationship between neuronal loss and visual sensitivity decline, whereas persistent discrepancies underscore individual variability and current methodological constraints. Taken together, these findings advocate for a transition from static classification toward continuous longitudinal monitoring of both structural and functional parameters. Integration of imaging biomarkers with functional assessments and emerging technologies holds promise for improving early detection, refining risk stratification, and optimizing management strategies in individuals with suspected glaucomatous optic neuropathy.

DECLARATION

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Ethical Approval: This study is a review based on previously published data; therefore, ethical approval was not required. Informed

Data Availability: All data analyzed during this study are included in the published articles referenced within this manuscript. Additional details are available from the corresponding author upon reasonable request.

Author Contributions: All authors contributed significantly to the conception, design, data collection, analysis, and interpretation of the study. All authors were involved in drafting and revising the manuscript critically for important intellectual content and approved the final version for publication.

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REFERENCES

1. Weinreb RN, Aung T, Medeiros FA. The pathophysiology and treatment of glaucoma. Lancet. 2014;383(9912):1901–1912.
2. Tham YC, Li X, Wong TY, Quigley HA, Aung T, Cheng CY. Global prevalence of glaucoma. Ophthalmology. 2014;121(11):2081–2090.
3. Medeiros FA, Zangwill LM, Bowd C, Mansouri K, Weinreb RN. The structure and function relationship in glaucoma. Prog Retin Eye Res. 2012;31(2):174–192.
4. Quigley HA, Addicks EM, Green WR. Optic nerve damage in glaucoma. Arch Ophthalmol. 1982;100(1):135–146.
5. Hood DC, Kardon RH. A framework for comparing structural and functional measures. Prog Retin Eye Res. 2007;26(6):688–710.
6. Chauhan BC, Garway-Heath DF, Goñi FJ, et al. Practical recommendations for measuring rates of visual field change. Invest Ophthalmol Vis Sci. 2008;49(10):4386–4394.
7. Leung CK, Yu M, Weinreb RN, et al. Retinal nerve fiber layer imaging. Ophthalmology. 2012;119(4):731–737.
8. Mwanza JC, Durbin MK, Budenz DL. Interocular symmetry in RNFL thickness. Ophthalmology. 2011;118(12):2404–2411.
9. Hood DC, Raza AS, de Moraes CG, et al. Initial damage in glaucoma. Prog Retin Eye Res. 2013;32:1–21.
10. Medeiros FA, Lisboa R, Weinreb RN, et al. Predicting functional loss in glaucoma. Ophthalmology. 2012;119(9):1809–1815.
11. Zangwill LM, Weinreb RN, Berry CC, et al. Confocal scanning laser ophthalmoscopy. Arch Ophthalmol. 2001;119(10):1492–1499.
12. Burgoyne CF. A biomechanical paradigm for glaucomatous damage. Exp Eye Res. 2011;93(2):120–132.
13. Harwerth RS, Carter-Dawson L, Shen F, et al. Ganglion cell losses underlying visual field defects. Invest Ophthalmol Vis Sci. 1999;40(10):2242–2250.

14. Leung CK, Ye C, Weinreb RN, et al. Retinal nerve fiber layer imaging with OCT. Ophthalmology. 2010;117(2):267–274.
15. Garway-Heath DF, Ruben ST, Viswanathan A, et al. Mapping structure to function. Invest Ophthalmol Vis Sci. 2000;41(7):1809–1816.
16. Sommer A, Katz J, Quigley HA, et al. Clinically detectable nerve fiber loss. Arch Ophthalmol. 1991;109(1):77–83.
17. Caprioli J, Prum B, Zeyen T. Rates of progression in glaucoma. Am J Ophthalmol. 2009;147(3):411–418.
18. Budenz DL, Fredette MJ, Feuer WJ, Anderson DR. Reproducibility of RNFL thickness. Ophthalmology. 2008;115(4):661–666.
19. Leung CK, Cheung CY, Weinreb RN, et al. RNFL imaging with spectral-domain OCT. Ophthalmology. 2009;116(7):1257–1263.
20. Hood DC, De Moraes CG. Challenges to the structure–function relationship. Prog Retin Eye Res. 2018;67:1–14.
21. Chauhan BC, O'Leary N, AlMobarak FA, et al. Enhanced detection of progression. Ophthalmology. 2014;121(9):1583–1592.
22. Tatham AJ, Medeiros FA. Detecting structural progression in glaucoma. Ophthalmology. 2017;124(12 Suppl):S57–S65.
23. Medeiros FA, Alencar LM, Zangwill LM, et al. Prediction of visual field progression. Invest Ophthalmol Vis Sci. 2009;50(4):1675–1681.
24. Banister K, Boachie C, Bourne R, et al. Can automated imaging replace visual field testing. Br J Ophthalmol. 2016;100(7):843–848.
25. Yu M, Lin C, Weinreb RN, et al. Risk of progression in glaucoma suspects. Ophthalmology. 2014;121(11):2189–2196.
26. De Moraes CG, Juthani VJ, Liebmann JM, et al. Risk factors for visual field progression. Ophthalmology. 2011;118(4):755–761.
27. Mwanza JC, Oakley JD, Budenz DL, et al. Macular ganglion cell analysis. Invest Ophthalmol Vis Sci. 2011;52(11):8323–8329.
28. Oddone F, Centofanti M, Rossetti L, et al. Exploring the glaucoma structure–function relationship. Eye. 2016;30(1):1–6.
29. Tan O, Li G, Lu AT, et al. Mapping of macular ganglion cells. Ophthalmology. 2008;115(6):949–956.
30. Rao HL, Pradhan ZS, Weinreb RN, et al. Structure-function relationship in glaucoma. Invest Ophthalmol Vis Sci. 2014;55(9):5419–5426.
31. Sung KR, Kim JS, Wollstein G, et al. Imaging of the macular ganglion cell layer. Ophthalmology. 2012;119(9):1915–1922.
32. Medeiros FA, Zangwill LM, Alencar LM, et al. Detection of progression with OCT. Invest Ophthalmol Vis Sci. 2009;50(4):1676–1684.
33. Hood DC, Fortune B, Arthur SN, et al. Blood vessel contributions to RNFL thickness. Invest Ophthalmol Vis Sci. 2008;49(8):3119–3126.
34. Leung CK, Cheung CY, Weinreb RN. Evaluation of retinal nerve fiber layer progression. Ophthalmology. 2011;118(8):1558–1562.
35. Lisboa R, Leite MT, Zangwill LM, et al. Diagnosing preperimetric glaucoma. Ophthalmology. 2012;119(11):2261–2269.
36. Chauhan BC, Burgoyne CF. From clinical examination to clinical trials. Prog Retin Eye Res. 2013;37:1–21.
37. Medeiros FA, Weinreb RN. Evaluation of progressive optic disc change. Ophthalmology. 2012;119(6):1126–1132.
38. Bowd C, Zangwill LM, Berry CC, et al. Detecting glaucoma progression. Invest Ophthalmol Vis Sci. 2001;42(2):351–359.
39. Weinreb RN, Khaw PT. Primary open-angle glaucoma. Lancet. 2004;363(9422):1711–1720.
40. Quigley HA. Glaucoma. Lancet. 2011;377(9774):1367–1377.
41. Hood DC, Raza AS. On improving the use of OCT imaging. Prog Retin Eye Res. 2014;39:1–27.
42. Acharya A, Chowdhury P, Shah B. Effectiveness of low vision rehabilitation services on visual function and quality of life in individuals with visual impairment: A systematic review. New Era Med J. 2025;1(2):1–8.

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