

Structural and Functional Retinal and Optic Nerve Changes in Parkinson's Disease: An OCT-Based Study

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Running Title: OCT Findings in Parkinson's Disease

Abstract

Background: Parkinson's disease (PD) is characterized by progressive neurodegeneration involving not only motor circuits but also visual pathways, including retinal and optic nerve structures. Vision is one of the non-motor systems altered in PD, and patients often experience decreased visual acuity, contrast sensitivity, and color vision. Optical coherence tomography (OCT) is a non-invasive method for evaluating these structures and may disclose early changes. **Aim:** This study used OCT and visual function tests to analyze retinal and optic nerve changes in Parkinson's patients compared with controls. **Methods:** This comparative cross-sectional study included 16 Parkinson's patients (32 eyes) and 16 age- and sex-matched controls (32 eyes). OCT assessed the peripapillary retinal nerve fiber layer (pRNFL), macular RNFL (mRNFL), ganglion cell layer plus inner plexiform layer (GCL+), ganglion cell complex (GCL++), and optic disc characteristics. Functional tests included best-corrected visual acuity (BCVA), contrast sensitivity, and color vision. **Results:** Compared with controls, Parkinson's patients showed significant thinning of the mRNFL ($p = 0.012$) and GCL++ ($p = 0.029$), along with optic disc alterations including a larger cup-to-disc ratio ($p < 0.001$). Inferior quadrant RNFL thinning was also observed in the PD group ($p < 0.001$). Patients demonstrated reduced BCVA ($p < 0.001$), lower contrast sensitivity ($p < 0.01$), and impaired color vision ($p < 0.001$). **Conclusion:** Parkinson's disease is associated with inner-retinal thinning and visual dysfunction. OCT combined with visual testing may provide non-invasive markers of PD-related neurodegeneration. These findings highlight the potential of OCT as an adjunctive biomarker, although larger studies are needed for validation.

Keywords: Optical coherence tomography, retinal nerve fiber layer, ganglion cell complex, visual function, optic nerve atrophy

Introduction

Parkinson's disease is a neurodegenerative disorder involving dopaminergic neuron loss in the basal ganglia. While known for motor symptoms, it also affects the visual pathway⁽¹⁾. Degenerative retinal changes, reflected by thinning of both the retinal nerve fiber layer (RNFL) and ganglion cell layers, may reflect central nervous system damage visible through retinal imaging⁽²⁾.

Optical coherence tomography (OCT), a non-invasive, high-resolution imaging technique, allows for thorough assessment of retinal and optic nerve components. Multiple studies consistently report RNFL and ganglion cell thinning, along with optic disc changes in Parkinson's disease, highlighting OCT as a promising candidate biomarker⁽³⁾.

This study was conducted to investigate structural and functional retinal

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abnormalities in idiopathic Parkinson's disease using OCT and visual tests and to compare findings with matched healthy controls. It also explored the relationship between OCT parameters and disease severity assessed through the Montreal Cognitive Assessment (MoCA) and Unified Parkinson's Disease Rating Scale (UPDRS). This study provides novel data from an Egyptian cohort, representing an under-studied North African population, and uniquely combines OCT-derived structural measures with visual function to offer a comprehensive assessment of PD-related neurodegeneration.

Subjects/Materials and Methods

Study Design and Participants

The research was designed as a cross-sectional comparison between groups was carried out at Suez Canal University Hospitals from June 2025 to September 2025. The study involved two groups: The first group included 16 patients (32 eyes) clinically diagnosed with idiopathic Parkinson's disease (PD) diagnosed by a movement-disorders neurologist according to MDS clinical diagnostic criteria.

The second group consisted of 16 age- and gender-matched healthy controls (32 eyes) with no history of neurological or ophthalmologic disorders. Controls were chosen to have demographic features similar to those of the Parkinson's group.

Inclusion Criteria

- Age between 60 and 75 years
- Both sexes were included
- Parkinsonian diagnosis (idiopathic)

Exclusion Criteria

- History of any optic nerve disease (e.g., glaucoma)

- History of retinal disease (e.g., diabetic retinopathy)
- Significant refractive error ($> \pm 6.00$ diopters spherical equivalent)
- History of ocular trauma or surgery
- Media opacities, poor fixation that may disrupt OCT or visual function testing.
- Patients with atypical parkinsonian syndromes
- Patients with systemic or neurological disorders that may affect retinal structure or OCT interpretation (e.g., Alzheimer's disease, chronic obstructive pulmonary disease, chronic kidney failure, or other neurodegenerative diseases).

Ophthalmic examination:

Each participant received a comprehensive ophthalmic evaluation, including slit-lamp biomicroscopy, dilated fundus assessment, and measurement of intraocular pressure using Goldmann applanation tonometry Shin Nippon applanation tonometer (Japan).

OCT Imaging Protocol:

Spectral-domain OCT (Topcon Maestro2, Topcon Corp., Tokyo, Japan) acquired a 3.4-mm peripapillary circle scan (average and quadrant RNFL) and a 6×6-mm macular cube (GCL+, GCL++, and macular RNFL) centered on the fovea using ETDRS-style grids (parafoveal 1–3 mm; perifoveal 3–6 mm). Scans were accepted only if they met the device quality index threshold and passed manual segmentation review by a masked grader; images with motion artifact, decentration, or segmentation error were repeated and excluded if unresolved.

Optic Nerve Functional Testing:

Best corrected visual acuity (BCVA) was assessed using a Snellen chart and

converted to Log MAR for analysis. Contrast sensitivity was assessed with the Pelli-Robson chart, based on the last three triplets with ≥ 2 correct letters. Color vision was evaluated using the 21 diagnostic plates of the 38-plate Ishihara test. All assessments were conducted by a single examiner blinded to group assignment.

Neurological Examination:

All Parkinson's patients had comprehensive clinical data recorded, including disease duration, time since diagnosis, diagnostic delay (from symptom onset to diagnosis), treatment regimen (levodopa and adjuncts), chronic systemic illness, and MoCA score. Motor severity was measured using the UPDRS. Disease was classified according to age of onset and phenotype (e.g., tremor-dominant, akinetic-rigid).

These characteristics were examined for relationships with OCT-derived retinal alterations, specifically RNFL and ganglion cell complex thickness, to see whether disease chronicity, systemic illness, cognitive decline, or motor severity influenced optic nerve degeneration.

Before participating, each individual provided written informed consent. The Research Ethics Committee of the Faculty of Medicine at Suez Canal University examined and approved the work on May 26, 2025 (Research 5848#). All study procedures followed the ethical criteria outlined in the Declaration of Helsinki.

Statistical Analysis:

Statistical analyses were conducted using SPSS version 25.0 (IBM Corp., Armonk, NY, USA). The Shapiro–Wilk test was applied to

evaluate normality. Depending on data distribution, group differences were assessed using independent-samples t-tests or Mann–Whitney U tests, while categorical variables were compared with χ^2 or Fisher's exact tests. Relationships between OCT measurements and MoCA/UPDRS scores were examined using Spearman correlation coefficients. Results are reported as exploratory, including effect sizes and 95% confidence intervals where relevant. A p-value < 0.05 was considered statistically significant.

Results

Demographic characteristics

The study included 64 eyes of 32 participants in total: 16 patients with a clinical diagnosis of Parkinson's disease (9 males, 7 females; mean age: 67.4 ± 4.6 years) and 16 healthy controls who were matched for age and sex. All participants underwent functional testing of the optic nerve and OCT imaging.

OCT Structural Findings:

Retinal structural analysis demonstrated multiple significant differences between the groups (**Table 1**). The average peripapillary RNFL thickness did not differ significantly ($98.3 \pm 7.6 \mu\text{m}$ in Parkinson's disease vs. $96.8 \pm 10.9 \mu\text{m}$ in controls, $p = 0.472$), suggesting that global RNFL integrity may be relatively preserved in early disease. However, the Parkinson's disease group showed a significantly thinner inferior quadrant RNFL ($108.7 \pm 9.9 \mu\text{m}$) compared to controls ($134.5 \pm 11.5 \mu\text{m}$, $p < 0.001$).

Table 1: Comparison of Optical Coherence Tomography (OCT) Structural Parameters Between Parkinsonism Patients and Healthy Controls.

Parameter	Parkinson's disease Mean $\pm$ SD	Control Mean $\pm$ SD	p- value	FDR- adjusted p	Significant after FDR?
Average RNFL (μm)	98.3 $\pm$ 7.6	96.8 $\pm$ 10.9	0.472	0.506	No
Superior RNFL (μm)	110.0 $\pm$ 6.9	105.3 $\pm$ 12.1	0.118	0.148	No
Inferior RNFL (μm)	108.7 $\pm$ 11.5	134.5 $\pm$ 9.9	<0.001	0.003	Yes
Nasal RNFL (μm)	83.1 $\pm$ 14.6	84.2 $\pm$ 10.4	0.763	0.763	No
Temporal RNFL (μm)	65.9 $\pm$ 9.2	69.3 $\pm$ 8.7	0.185	0.214	No
Macular RNFL (mRNFL, μm)	39.7 $\pm$ 10.2	46.1 $\pm$ 9.5	0.012	0.023	Yes
GCL+ (μm)	59.1 $\pm$ 6.8	65.9 $\pm$ 15.3	0.074	0.101	No
GCL++ (μm)	92.3 $\pm$ 7.5	97.6 $\pm$ 5.2	0.029	0.044	Yes
Disc area (mm^2)	2.09 $\pm$ 0.77	2.60 $\pm$ 0.33	0.001	0.003	Yes
Rim area (mm^2)	1.69 $\pm$ 0.85	2.10 $\pm$ 0.34	0.024	0.040	Yes
Cup area (mm^2)	0.37 $\pm$ 0.11	0.18 $\pm$ 0.05	<0.01	0.021	Yes
C/D Ratio	0.42 $\pm$ 0.11	0.32 $\pm$ 0.08	<0.001	0.003	Yes

Data are presented as mean $\pm$ standard deviation. RNFL: retinal nerve fiber layer; GCL+: ganglion cell layer plus inner plexiform layer; GCL++: GCL+ plus RNFL; mRNFL: macular retinal nerve fiber layer; C/D: cup-to-disc. $p < 0.05$ indicates statistical significance.

Parkinson's patients had a thinner temporal quadrant RNFL (65.9 $\pm$ 9.2 μm) than controls (69.3 $\pm$ 8.7 μm), although the difference was not statistically significant

($p = 0.185$). Similarly, no significant differences were identified in the superior (110.0 $\pm$ 6.9 μm vs. 105.3 $\pm$ 12.1 μm) with $p = 0.118$ or nasal quadrants (83.1 $\pm$ 14.6 μm vs. 84.2 $\pm$ 10.4 μm) with $p = 0.763$.

Parkinson's group had significantly lower mRNFL (39.7 $\pm$ 10.2 μm) than controls (46.1 $\pm$ 9.5 μm) with $p = 0.012$, indicating early neurodegenerative involvement in central retinal areas, as illustrated in Figure 1.

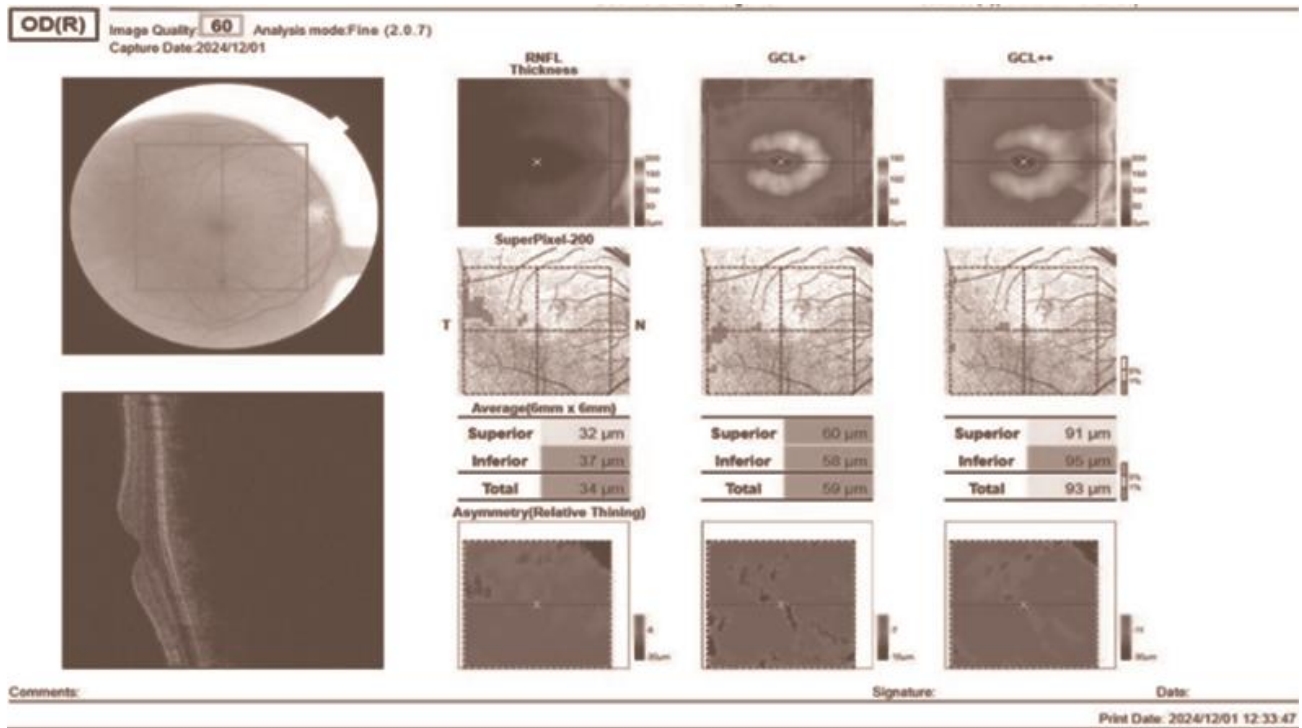


Figure 1: Macular OCT of the right eye in a patient with Parkinson's disease: shows reduced macular RNFL thickness (Total: 34 μ m), particularly in the superior quadrant (32 μ m), indicating inner retinal thinning. GCL+ and GCL++ layers show relative thinning.

Parkinson's patients had a significantly lower GCL++ thickness ($92.3 \pm 7.5 \mu$ m vs. $97.6 \pm 5.2 \mu$ m) with $p = 0.029$, while GCL+ thickness showed a trend toward thinning ($59.1 \pm 6.8 \mu$ m vs. $65.9 \pm 15.3 \mu$ m) with $p = 0.074$, but this did not achieve statistical significance.

The morphology of the optic disc was also altered. Parkinson's patients had a significantly decreased disc area ($2.09 \pm 0.77 \text{ mm}^2$) compared to controls ($2.60 \pm 0.33 \text{ mm}^2$) with $p = 0.001$. The Parkinson's group had a smaller rim area ($1.69 \pm 0.85 \text{ mm}^2$ vs. $2.10 \pm 0.34 \text{ mm}^2$) with $p = 0.024$. Parkinson's patients had a larger cup area ($0.37 \pm 0.11 \text{ mm}^2$ vs. $0.18 \pm 0.05 \text{ mm}^2$) with $p < 0.01$ and a considerably increased cup-to-disc (C/D) ratio (0.42 ± 0.11) compared to controls (0.32 ± 0.08) with $p < 0.001$, consistent with larger optic disc cupping, which may reflect structural change but is not diagnostic of glaucoma.

After applying FDR correction ($q = 0.05$), significant differences persisted for inferior RNFL, macular RNFL, GCL++, disc area, rim area, cup area, and cup-to-disc ratio (all FDR-adjusted $p < 0.05$). GCL+, average RNFL, and other quadrant-specific RNFL measures did not survive correction.

Optic Nerve Functional Findings

Optic nerve functions were assessed in both groups and showed significant visual impairment in Parkinson's group as presented in Table 2. BCVA, measured in Log MAR, showed a significant reduction in the Parkinson's group (0.18 ± 0.09) than in healthy controls (0.06 ± 0.04 , $p < 0.001$), suggesting decreased central visual clarity. Contrast sensitivity was also assessed in both groups. It was significantly reduced in Parkinson's group (1.50 ± 0.18 log units) with comparison to controls (1.73 ± 0.11 log units, $p < 0.01$), indicating decreased ability to perceive low-contrast details which is a

common early visual impairment in Parkinsonian syndromes.

Furthermore, assessment of color vision revealed a statistically significant decrease in Parkinson's patients, with an average score of 16.4 ± 3.1 correctly identified plates compared to 20.8 ± 0.6 in controls ($p < 0.001$). This confirms the existence of

modest red-green dyschromatopsia, which is in line with table 2's depiction of dopaminergic retinal pathway impairment. After FDR correction, all three functional measures (BCVA, contrast sensitivity, and color vision) remained statistically significant (all FDR-adjusted $p < 0.05$).

Table 2: Comparison of Optic Nerve Functional Parameters Between Parkinson's disease Patients and Healthy Controls.

Functional Parameter	Parkinson's disease Mean $\pm$ SD	Control Mean $\pm$ SD	p-value	FDR-adjusted p	Significant after FDR?
BCVA (LogMAR)	0.18 ± 0.09	0.06 ± 0.04	<0.001	0.003	Yes
Contrast Sensitivity (log)	1.50 ± 0.18	1.73 ± 0.11	<0.01	0.021	Yes
Color Vision (Ishihara)	16.4 ± 3.1 plates	20.8 ± 0.6 plates	<0.001	0.003	Yes

Data are presented as mean $\pm$ standard deviation. BCVA: best-corrected visual acuity in LogMAR; contrast sensitivity measured in log units; color vision assessed by the Ishihara 38-plate test (maximum score = 21). $p < 0.05$ indicates statistical significance.

Characteristics of Parkinson's disease Patients, Table 3.

The mean duration of Parkinson's symptoms was 4.1 ± 2.3 years, with an average diagnosis delay of 1.4 ± 1.1 years.

The average time since formal diagnosis is 2.7 ± 1.9 years. The majority of patients had late-onset Parkinson's disease, with 65% taking a combination of levodopa, dopamine agonists, and MAO-B inhibitors. Chronic illness was reported by 45% of the participants. The average MoCA score was 23.4 ± 2.8 , suggesting mild cognitive impairment. The mean UPDRS total score was 73.8 ± 18.5 , reflecting the overall motor severity of the disease.

Table 3: Clinical Characteristics of Parkinson's disease Patients (n = 16)

Parameter	Mean $\pm$ SD / %
Duration of Parkinsonism symptoms (years)	4.1 ± 2.3
Diagnostic delay (years)	1.4 ± 1.1
Time since formal diagnosis (years)	2.7 ± 1.9
Montreal Cognitive Assessment (MoCA) score	23.4 ± 2.8
Unified Parkinson's Disease Rating Scale (UPDRS)	73.8 ± 18.5
Late-onset Parkinson's disease	Majority (qualitative)
On multi-agent regimen (e.g., L-Dopa + adjuncts)	65%
Chronic systemic illness	45%

Exploratory correlation analysis revealed several trends, Figure 2:

- Longer duration of disease and delayed diagnosis were linked to thinner GCL++ and reduced macular

RNFL, indicating neuroretinal degeneration with chronicity.

- Lower MoCA scores were found to have a weak-to-moderate correlation with temporal RNFL thinning, suggesting neurodegeneration in both visual and cognitive pathways.
- Patients with concomitant chronic illnesses had slightly smaller disc

areas and higher cup-to-disc ratios, but these findings were not statistically significant.

- No significant correlations were found between PD categorization subtype and OCT characteristics. However, akinetic-rigid types had thinner RNFLs overall.

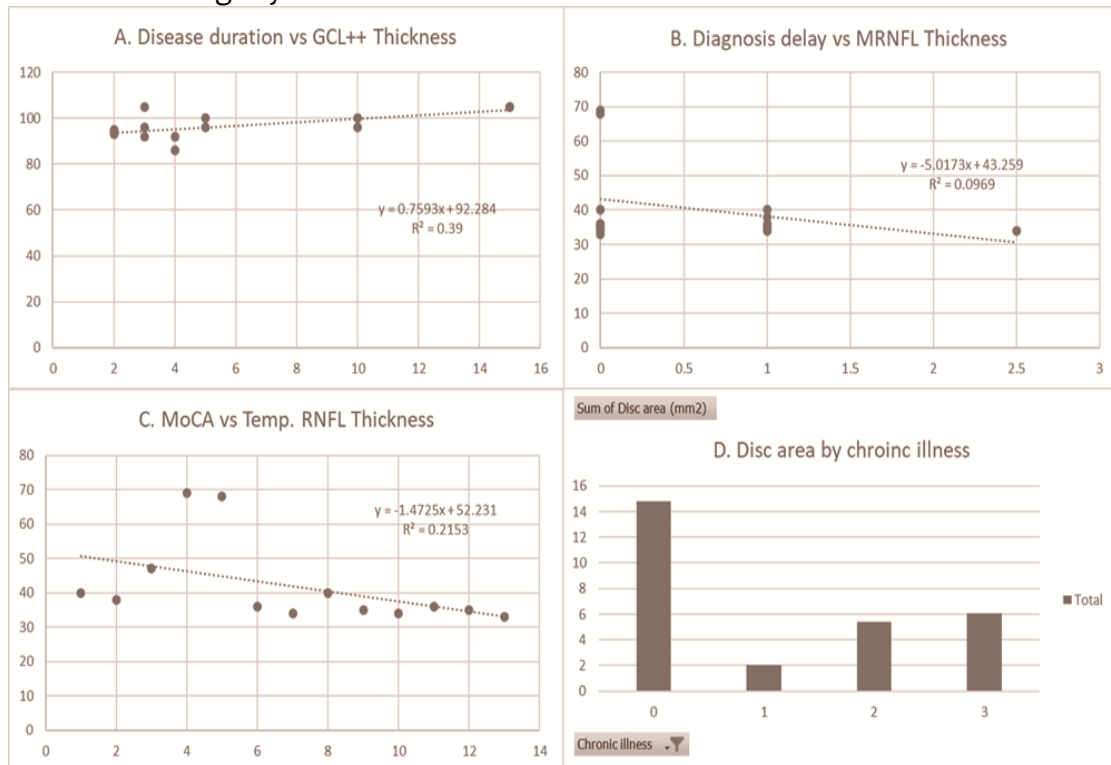


Figure 2: Exploratory Correlation Trends in PD (N=16).

These data support a multifaceted impact on retinal integrity in Parkinson's disease, which includes both neurological and systemic illness variables. Exploratory correlations with UPDRS and MoCA did not reach statistical significance (all $p > 0.1$), though weak, non-significant trends were observed in the same direction as prior studies.

Discussion

Our findings support inner-retinal involvement in idiopathic PD, with thinner macular layers (mRNFL, GCL++) and measurable decrements in visual acuity, contrast sensitivity, and color discrimination. Average peripapillary RNFL was similar between groups, aligning with reports that macular measures may be more sensitive to early disease. While El-Kattan et al. have previously reported RNFL thinning in Egyptian Parkinson's

patients, our study extends this work by integrating macular GCL++, disc morphometry, and visual functional tests (BCVA, contrast sensitivity, color vision) alongside exploratory correlations with MoCA and UPDRS⁽⁴⁾. This provides a more comprehensive picture of PD-related neurodegeneration.

The higher C/D ratio in PD should be interpreted cautiously as a structural index rather than evidence of glaucoma, given normal IOP screening and the influence of ocular biometry. Importantly, inferior quadrant RNFL was significantly thinner in the PD group, consistent with prior reports that inferior and temporal peripapillary axons may be preferentially vulnerable in PD^(2,3). This localized thinning may represent an early structural biomarker of visual pathway involvement and warrants confirmation in larger, longitudinal studies. Taken together, these results suggest that Parkinson's disease is associated with inner-retinal alterations and visual dysfunction. Optical coherence tomography (OCT) combined with visual testing may provide a non-invasive marker of PD-related neurodegeneration, although confirmation in larger cohorts is needed.

GCL++ was significantly reduced, while GCL+ showed no significant difference. This differential likely reflects the wider tissue captured by GCL++, making it more sensitive to combined ganglion-cell and axonal loss, whereas GCL+ may miss subtle, early changes. Methodological factors (segmentation differences across scans), normal anatomical variability, and the modest sample size may also contribute.

The preferential thinning of the RNFL and GCL in the inferior and temporal quadrants may reflect selective involvement of the papillomacular bundle, which carries fibers

responsible for central vision. These regions have a high density of metabolically active ganglion cells that could be more sensitive to dopaminergic deficiency or retrograde degeneration from higher visual centers in Parkinson's disease. Vascular or mitochondrial factors may also contribute to this localized vulnerability.

We observed greater RNFL thinning in akinetic-rigid than tremor-dominant phenotypes, but this difference was not statistically significant—likely due to limited power. Larger, stratified cohorts are needed to determine whether OCT can reliably distinguish PD motor subtypes. Macular RNFL and GCL++ thinning, along with optic disc alterations, echo Satue et al.'s observation of early neurodegenerative indicators⁽²⁾.

Temporal RNFL thinning was not significant in our cohort but remains a known feature of Parkinsonian optic neuropathy⁽³⁾. Interestingly, inferior RNFL thickness was significantly decreased in our Parkinson's group, a finding consistent with Garcia-Martin et al.⁽³⁾. Possible explanations include retrograde trans-synaptic neuroaxonal loss, segmentation artefact, or measurement variability; longitudinal imaging is required to clarify this observation.

Our results support prior evidence that inner-retinal thinning may reflect retrograde trans-synaptic degeneration from cortical or subcortical injury^(2,5,6). Garcia-Martin et al. showed that Fourier-domain OCT detects early GCL and RNFL alterations⁽³⁾, while El-Kattan et al. found RNFL thinning correlated with disease severity in an Egyptian cohort⁽⁵⁾.

Functionally, patients had worse BCVA, reduced contrast sensitivity, and impaired color vision, consistent with Price et al.⁽⁷⁾,

who attributed such deficits to dopaminergic retinal degeneration, and with Archibald et al.⁽⁸⁾, who emphasized higher-order cortical contributions. Contrast sensitivity loss, in particular, has been highlighted as an early marker of visual pathway involvement by Nowacka et al.⁽⁹⁾.

In our exploratory analysis, participants with lower MoCA results tended to have thinner temporal RNFL values, indicating that cognitive decline may occur alongside subtle retinal changes. Likewise, patients who scored higher on the UPDRS, reflecting more severe motor symptoms, often showed reduced thickness in macular and GCL++ layers. The direction of these associations is consistent with prior reports by Satue et al.⁽²⁾ and Chang et al.⁽¹⁰⁾, which linked retinal thinning to cognitive decline. While our sample size limits definitive modeling, these results reinforce the plausibility of OCT as a marker of cognitive and motor burden in PD.

Finally, optic disc differences, including larger C/D ratios, were evident in Parkinson's patients. While these findings echo Garcia-Martin et al.'s report of disc metrics as potential biomarkers⁽³⁾, they should be interpreted cautiously, as disc parameters are influenced by ocular biometry and segmentation variability.

The main limitations of this study are the modest sample size and cross-sectional design, which limit statistical power and preclude causal inference.

Conclusion

Optical coherence tomography (OCT) is an effective, non-invasive tool for detecting retinal and optic nerve alterations in Parkinson's disease. RNFL and GCL thinning, particularly in the inferior and temporal quadrants, may represent

potential surrogate markers of neurodegeneration in both clinical and research settings. Our findings support incorporating OCT-derived structural measures into comprehensive Parkinson's disease evaluation frameworks, with the potential to complement cognitive and motor assessments. However, these results are preliminary and require confirmation in larger longitudinal studies before OCT can be considered predictive at the individual level.

List of Abbreviations

Abbreviation	Full Term
BCVA	Best-corrected visual acuity
C/D	Cup-to-disc ratio
CI	Confidence interval
CS	Contrast sensitivity
FDR	False discovery rate
GCL	Ganglion cell layer
GCL+	Ganglion cell layer plus inner plexiform layer
GCL++	Ganglion cell complex (GCL+ plus RNFL)
INL	Inner nuclear layer
IPL	Inner plexiform layer
IOP	Intraocular pressure
LogMAR	Logarithm of the minimum angle of resolution
MoCA	Montreal Cognitive Assessment
OCT	Optical coherence tomography
ONL	Outer nuclear layer
OPL	Outer plexiform layer
PD	Parkinson's disease
p	Probability value
RNFL	Retinal nerve fiber layer
SD	Standard deviation
SPSS	Statistical Package for the Social Sciences
UPDRS	Unified Parkinson's Disease Rating Scale
VA	Visual acuity
VEP	Visual evoked potential

Ethics approval & consent

Approval for the study was granted by the Departmental Research Committee and the Research Ethics Committee of the Faculty of Medicine, Suez Canal University on May 26, 2025 (Reference: Research 5848#). All procedures adhered to the ethical principles outlined in the Declaration of Helsinki, and written informed consent was obtained from all participants prior to being included in the study.

Conflicts of Interest:

The authors disclose no financial or conflicting interests that are relevant to this work.

Author Contributions:

E.M.M. was the principal author, corresponding author, and the sole ophthalmologist involved in this study. He designed and coordinated the ophthalmologic component of the research, performed all comprehensive eye examinations, acquired and analyzed the OCT data, and led the interpretation of the ophthalmic findings. He also conducted the statistical analysis and was responsible for writing the entire manuscript, including figure preparation and final critical revisions.

M.E.A., B.H.F., A.S.A., and E.A.H. all neurologists helped with patient recruitment, clinical examination, Parkinsonism diagnosis, and adherence to diagnostic criteria. They also facilitated communication between the neurology and ophthalmology departments, assisted with data collection, and contributed to the literature review and manuscript revision. In addition, **E.A.H** ensured the neurological assessments were conducted consistently and aligned with established clinical rating scales and diagnostic frameworks.

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Data availability:

Access to the datasets collected or analyzed during this work may be granted by the corresponding author upon a valid request and in accordance with ethical rules.

References

1. Bodis-Wollner I. Retinopathy in Parkinson disease. *J Neural Transm.* 2009;116(11):1493–1501.
2. Satue M, Seral M, Otin S, Alarcia R, et al. Retinal thinning and correlation with functional disability in patients with Parkinson's disease. *Br J Ophthalmol.* 2014;98(3):350–355.
3. Garcia-Martin E, Satue M, Fuertes I, et al. Ability and reproducibility of Fourier-domain optical coherence tomography to detect retinal nerve fiber layer atrophy in Parkinson's disease. *Ophthalmology.* 2012;119(10):2161–2167.
4. El-Kattan MM, Esmat SM, Esmail EH, Deraz HA, Ismail RS. Optical coherence tomography in patients with Parkinson's disease. *Egypt J Neurol Psychiatry Neurosurg.* 2022; 58:21. 1
5. Svetel M, Marić G, Božić M, et al. Retinal thickness in patients with Parkinson's disease and Dopa-responsive dystonia—Is there any difference? *Biomedicines.* 2025;13(5):1227.
6. Murueta-Goyena A, Teijeira-Portas S, Blanco Martín E, et al. Dynamics of retinal changes in early-stage Parkinson's disease. *Acta Neuropathol Commun.* 2025;13(1):20.
7. Price MJ, Feldman RG, Adelberg D, Kayne H. Abnormalities in color vision and contrast sensitivity in Parkinson's disease. *Neurology.* 1992;42(4):887–890.

8. Archibald NK, Clarke MP, Mosimann UP, Burn DJ. The retina in Parkinson's disease. *Brain*. 2009;132(Pt 5):1128–1145.
9. Nowacka B, Lubinski W, Honczarenko K, Potemkowski A, Safranow K. Ophthalmological features of Parkinson disease. *Med Sci Monit*. 2014; 20:2243–2249.
10. Chang Z, Xie F, Li H, Yuan F, Zeng L, Shi L, et al. Retinal nerve fiber layer thickness and associations with cognitive impairment in Parkinson's disease. *Front Aging Neurosci*. 2022; 14:832768.