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EFFECT OF ANTIOXIDANT SUPPLEMENTATION ON OXIDATIVE STRESS AND RETINAL NEURODEGENERATION BIOMARKERS IN PATIENTS WITH PRIMARY OPEN-ANGLE GLAUCOMA: A RANDOMIZED CONTROLLED TRIAL

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ABSTRACT

Background:

Primary open-angle glaucoma (POAG) is a leading cause of irreversible blindness, characterized by progressive retinal ganglion cell loss despite adequate intraocular pressure (IOP) control. Increasing evidence suggests that oxidative stress contributes to retinal neurodegeneration in glaucoma. Antioxidant supplementation may provide a neuroprotective approach by reducing oxidative damage and preserving retinal structures.

Objective:

To evaluate the effect of oral antioxidant supplementation on oxidative stress biomarkers and retinal neurodegeneration parameters in patients with primary open-angle glaucoma.

Methods:

This prospective, open-label randomized controlled trial was conducted over six months at the Department of Ophthalmology, Services Hospital Lahore. Forty patients with mild to moderate primary

open-angle glaucoma (POAG) were randomly allocated to an antioxidant supplementation group (n=20) or a control group (n=20). The intervention group received oral antioxidants in addition to standard glaucoma treatment, while the control group received standard glaucoma treatment alone. Oxidative stress biomarkers (MDA, TAC, SOD, and GPx) and retinal structural parameters (RNFL and GCC thickness) were assessed at baseline and six months. Statistical significance was set at $p<0.05$.

Results:

All 40 participants completed the six-month follow-up. Baseline demographic, clinical, and glaucoma treatment characteristics were comparable between groups. The antioxidant group demonstrated a significant reduction in MDA levels (4.82 ± 0.72 to 3.71 ± 0.61 nmol/mL, $p<0.001$) with increased TAC, SOD, and GPx levels (all $p<0.001$). RNFL and GCC thickness remained stable in the antioxidant group, whereas the control group showed significant thinning of both parameters ($p<0.001$). No significant changes were observed in BCVA or IOP between groups. Pearson correlation analysis showed a negative correlation between MDA and RNFL thickness ($r=-0.52$, $p=0.001$), while TAC, SOD, and GPx showed positive correlations with retinal structural parameters.

Conclusion:

Antioxidant supplementation improved oxidative stress status and enhanced antioxidant defense mechanisms in patients with POAG. Preservation of RNFL and GCC thickness suggests a potential neuroprotective effect through reduction of oxidative stress-related retinal damage. These findings support antioxidant supplementation as a potential adjunctive strategy in glaucoma management, although further long-term studies with larger populations are required to evaluate its effect on disease progression.

Keywords: *Primary open-angle glaucoma, oxidative stress, antioxidants, retinal nerve fiber layer, ganglion cell complex*

Introduction

Primary open-angle glaucoma (POAG) is the most common form of glaucoma and remains a leading cause of irreversible blindness worldwide. [12] Despite substantial advances in intraocular pressure (IOP) lowering treatments, many patients continue to experience progressive retinal ganglion cell (RGC) loss and visual field deterioration even when IOP is adequately controlled. [1,2,4] This observation suggests that mechanisms beyond elevated IOP contribute to glaucomatous neurodegeneration and disease progression. [3]

Accumulating evidence indicates that oxidative stress plays a central role in the pathogenesis of POAG. Oxidative stress results from an imbalance between the production of reactive oxygen species (ROS) and the body's antioxidant defense systems, leading to cellular damage, mitochondrial dysfunction, inflammation, and apoptosis. In glaucoma, increased oxidative stress has been implicated in trabecular meshwork dysfunction, impaired aqueous humor outflow, optic nerve damage, and retinal ganglion cell degeneration. Several studies have demonstrated elevated levels of oxidative damage markers, including malondialdehyde (MDA), along with reduced antioxidant defense mechanisms such as superoxide dismutase (SOD), glutathione peroxidase (GPx), and total antioxidant capacity (TAC) in patients with glaucoma. [5,6,7,8,9]

Retinal neurodegeneration is a hallmark of POAG and is characterized by progressive loss of retinal ganglion cells and thinning of the retinal nerve fiber layer (RNFL) and ganglion cell complex (GCC). Spectral-domain optical coherence tomography (SD-OCT) has emerged as a reliable, non-invasive imaging modality for detecting and monitoring these structural changes. [10] Increasing evidence suggests that oxidative stress-mediated neuronal injury contributes significantly to retinal ganglion cell loss, providing a potential therapeutic target beyond conventional IOP reduction. [11, 12]

Antioxidant supplementation has attracted growing interest as a neuroprotective strategy in glaucoma. Nutritional antioxidants, including lutein, zeaxanthin, vitamins C and E, and zinc, have demonstrated antioxidant, anti-inflammatory, and neuroprotective properties in experimental and clinical studies. These compounds may reduce oxidative damage, enhance endogenous antioxidant defenses, preserve mitochondrial function, and potentially slow retinal neurodegeneration. [13, 14] However, although observational and experimental data support a beneficial role of antioxidants in ocular health, clinical

evidence evaluating their impact on biochemical oxidative stress markers and structural retinal outcomes in patients with POAG remains limited and inconclusive. Few randomized controlled trials have simultaneously evaluated oxidative stress biomarkers and retinal neurodegeneration in patients with primary open-angle glaucoma (POAG). Therefore, this study investigated the effect of oral antioxidant supplementation on serum oxidative stress biomarkers (MDA, TAC, SOD, and GPx) and retinal structural parameters (RNFL and GCC thickness) measured by SD-OCT over a six-month follow-up period.

METHODOLOGY

This prospective, open-label, randomized controlled trial with observer masking was conducted over a period of six months at the Department of Ophthalmology, Services Hospital Lahore, Pakistan, to evaluate the effect of oral antioxidant supplementation on oxidative stress biomarkers and retinal neurodegeneration parameters in patients with primary open-angle glaucoma (POAG). The sample size was calculated using G*Power version 3.1 software. Assuming an effect size of 0.90, a two-sided significance level of 0.05, 80% statistical power, and an allocation ratio of 1:1, the required sample size was 40 participants. Accordingly, 20 participants were assigned to the antioxidant supplementation group and 20 participants to the control group. [15] Patients aged 40–75 years with a confirmed diagnosis of mild to moderate POAG were recruited from the ophthalmology outpatient department. Diagnosis was based on characteristic glaucomatous optic nerve changes, retinal nerve fiber layer (RNFL) thinning on spectral-domain optical coherence tomography (SD-OCT), and an open anterior chamber angle confirmed by gonioscopy. [10, 16]

Eligible participants were required to have stable intraocular pressure (IOP) lowering treatment for at least three months before enrollment and no history of antioxidant supplementation during the preceding three months. Patients with secondary glaucoma, angle-closure glaucoma, glaucoma surgery or laser intervention within the previous six months, retinal diseases affecting OCT evaluation, other optic neuropathies, severe systemic illnesses affecting oxidative stress pathways, pregnancy, lactation, or anticipated poor compliance were excluded from the study. Participants were randomly allocated in a 1:1 ratio using a computer-generated randomization sequence. Allocation concealment was maintained through sequentially numbered opaque sealed envelopes prepared by an independent coordinator. Due to the nature of the intervention, participants and treating ophthalmologists were not blinded; however,

OCT assessors and laboratory personnel responsible for biochemical analyses remained masked to treatment allocation throughout the study.

All participants continued their prescribed IOP lowering therapy according to routine clinical management. Commonly used medications included latanoprost 0.005%, timolol 0.5%, and dorzolamide 2%, administered according to clinical requirements. Treatment regimens were documented at baseline and maintained throughout the study whenever clinically appropriate. Participants allocated to the intervention arm received one CYT Softgel capsule daily after breakfast and one Surbex Z tablet daily after lunch for six months in addition to standard glaucoma treatment. CYT Softgel contained lutein and zeaxanthin, while Surbex Z contained vitamin C, vitamin E, zinc, and B-complex vitamins. Participants in the control group received standard glaucoma treatment alone without antioxidant supplementation. Peripheral venous blood samples were collected at baseline, three months, and six months for assessment of malondialdehyde (MDA), total antioxidant capacity (TAC), superoxide dismutase (SOD), and glutathione peroxidase (GPx) using validated biochemical assay methods at an accredited research laboratory. [17, 18, 19, 20] Structural retinal assessment was performed using SD-OCT, and the evaluated parameters included average RNFL thickness and ganglion cell complex (GCC) thickness. Participants underwent comprehensive baseline evaluation. Follow-up assessments were performed at three and six months. Data were analyzed using IBM SPSS Statistics. Continuous variables were expressed as mean ± SD and categorical variables as frequencies and percentages. Normality was assessed using the Shapiro–Wilk test. Independent and paired t-tests, repeated measures ANOVA, Chi-square/Fisher's exact tests, and Pearson correlation analysis were applied as appropriate. A p-value <0.05 was considered statistically significant. Ethical approval was obtained from the institutional ethics committee, and written informed consent was obtained from all participants.

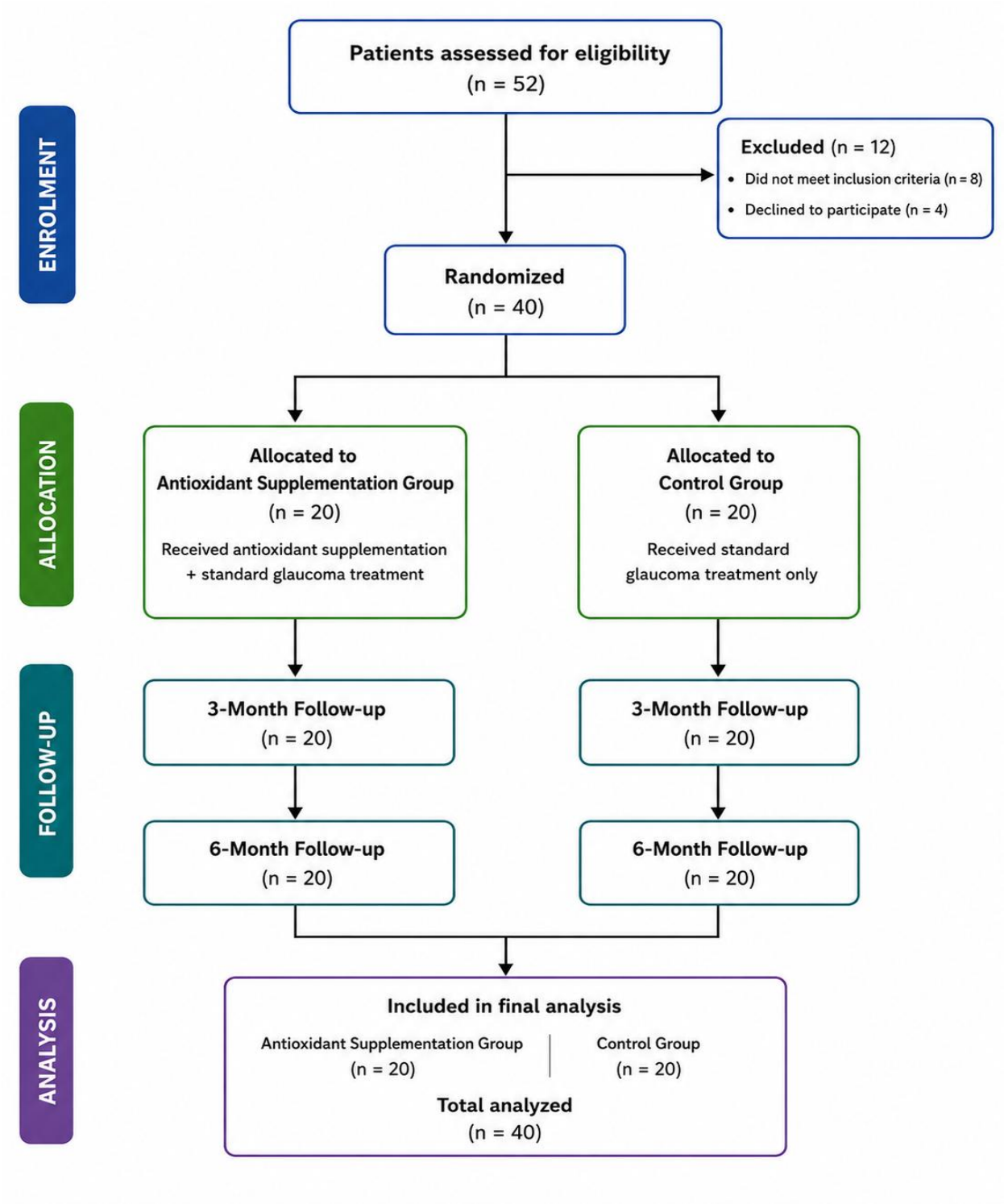

Figure 1: CONSORT Flow Chart [21]

RESULTS

Participant Demographic Characteristics

A total of 40 patients with primary open-angle glaucoma were enrolled and randomly allocated into two groups, with 20 participants in the antioxidant supplementation group receiving CYT Softgel and Surbex Z for 6 months and 20 participants in the control group continuing routine glaucoma therapy. All participants

completed the 6-month follow-up without any loss to follow-up. The mean age was 55.8 ± 7.4 years in the antioxidant group and 56.2 ± 6.9 years in the control group. Males represented 60% (n=12) of the antioxidant group and 55% (n=11) of the control group, while females accounted for 40% (n=8) and 45% (n=9), respectively. The demographic distribution was similar between both groups, indicating comparable baseline characteristics before intervention.

Table 1. Demographic Characteristics of Study Participants

Variable	Antioxidant Group (n=20)	Control Group (n=20)
Age (years), Mean ± SD	55.8 ± 7.4	56.2 ± 6.9
Gender		
Male, n (%)	12 (60%)	11 (55%)
Female, n (%)	8 (40%)	9 (45%)

Baseline Clinical Characteristics

The baseline clinical characteristics showed comparable disease profiles between the antioxidant and control groups. The mean duration of POAG was 4.3 ± 1.8 years in the antioxidant group and 4.5 ± 2.0 years in the control group. Mild POAG was present in 55% (n=11) of the antioxidant group and 50% (n=10) of the control group, while moderate POAG was observed in 45% (n=9) and 50% (n=10), respectively. Diabetes mellitus was reported in 25% (n=5) of the antioxidant group and 30% (n=6) of the control group, whereas hypertension was present in 40% (n=8) and 35% (n=7), respectively. Other systemic diseases were reported in 10% (n=2) of the antioxidant group and 15% (n=3) of the control group. Overall, both groups demonstrated similar baseline clinical characteristics before intervention.

Table 2. Baseline Clinical Characteristics of Study Participants

Variable	Antioxidant Group (n=20)	Control Group (n=20)
POAG duration (years), Mean ± SD	4.3 ± 1.8	4.5 ± 2.0
Mild POAG, n (%)	11 (55%)	10 (50%)
Moderate POAG, n (%)	9 (45%)	10 (50%)

Diabetes mellitus, n (%)	5 (25%)	6 (30%)
Hypertension, n (%)	8 (40%)	7 (35%)
Other systemic diseases, n (%)	2 (10%)	3 (15%)

Baseline Glaucoma Treatment Profile

The baseline glaucoma medication profile was comparable between the antioxidant and control groups. Latanoprost was used by 70% (n=14) of participants in the antioxidant group and 75% (n=15) in the control group. Timolol use was reported in 45% (n=9) and 40% (n=8) of participants, respectively, while dorzolamide was used by 30% (n=6) of the antioxidant group and 35% (n=7) of the control group. Multiple glaucoma medication use was observed in 40% (n=8) and 45% (n=9) of participants, respectively.

Table 3. Baseline Glaucoma Medication Characteristics

Medication	Antioxidant Group (n=20)	Control Group (n=20)
Latanoprost use	14 (70%)	15 (75%)
Timolol use	9 (45%)	8 (40%)
Dorzolamide use	6 (30%)	7 (35%)
Multiple glaucoma medications	8 (40%)	9 (45%)

Normality Assessment

The Shapiro–Wilk test was performed to evaluate the normal distribution of continuous variables before applying statistical analysis. All variables showed a normal distribution, including BCVA (p=0.238), IOP (p=0.421), RNFL thickness (p=0.142), GCC thickness (p=0.198), MDA (p=0.161), TAC (p=0.356), SOD (p=0.211), and GPx (p=0.249). Since all p-values were greater than 0.05, the data met the assumption of normality, and parametric tests including independent sample t-test, paired t-test, and repeated measures ANOVA were applied for further analysis.

Table 4. Shapiro-Wilk Normality Test for Continuous Variables

Variable	Statistic	p-value
BCVA	0.964	0.238
IOP	0.971	0.421
RNFL thickness	0.956	0.142
GCC thickness	0.961	0.198

MDA	0.958	0.161
TAC	0.969	0.356
SOD	0.962	0.211
GPx	0.965	0.249

Follow-up Clinical, Structural, and Biochemical Outcomes

Changes in clinical, OCT, and oxidative stress parameters were analyzed using Repeated Measures ANOVA and paired t-test during the 6-month follow-up period. BCVA remained stable in both groups, with no statistically significant changes observed in either the antioxidant or control group **(**$p>0.05$**),** indicating that supplementation maintained visual function without significant improvement or deterioration. IOP values also remained comparable throughout follow-up, with no significant changes observed ($p>0.05$), suggesting that antioxidant supplementation did not directly influence intraocular pressure. OCT analysis showed preservation of retinal structure in the antioxidant group, where RNFL thickness remained stable (right eye: 78.4 ± 6.8 to 77.9 ± 6.6 µm, $p=0.214$; left eye: 77.9 ± 7.0 to 77.5 ± 6.7 µm, $p=0.287$), whereas the control group showed significant RNFL reduction (right eye: 79.1 ± 7.1 to 75.4 ± 6.8 µm; left eye: 78.6 ± 6.5 to 74.9 ± 6.2 µm, $p<0.001$). Similarly, GCC thickness was preserved in the antioxidant group (right eye: 74.6 ± 5.9 to 74.2 ± 5.7 µm, $p=0.315$; left eye: 74.2 ± 6.1 to 73.8 ± 5.9 µm, $p=0.341$), while significant GCC thinning occurred in the control group ($p<0.001$). Oxidative stress analysis demonstrated significant improvement in the antioxidant group, with MDA levels decreasing from 4.82 ± 0.72 to 3.71 ± 0.61 nmol/mL ($p<0.001$), while TAC increased from 1.21 ± 0.18 to 1.68 ± 0.23 mmol/L, SOD increased from 82.5 ± 9.4 to 98.6 ± 11.1 U/mL, and GPx increased from 146.2 ± 18.5 to 175.4 ± 21.2 U/L (all $p<0.001$). The control group showed no significant improvement in antioxidant biomarkers. Overall, antioxidant supplementation was associated with reduced oxidative stress and preservation of retinal structural parameters, while BCVA and IOP remained stable during the follow-up period.

Table 5. Changes in Clinical, OCT, and Oxidative Stress Parameters During 6-Month Follow-up

Parameter	Group	Baseline	Month 3	Month 6	p-value

BCVA Right Eye (LogMAR)	**Antioxidant**	0.32 ± 0.15	0.31 ± 0.15	0.31 ± 0.16	0.684
	Control	0.31 ± 0.16	0.33 ± 0.17	0.35 ± 0.18	0.214
BCVA Left Eye (LogMAR)	**Antioxidant**	0.30 ± 0.14	0.30 ± 0.15	0.29 ± 0.15	0.731
	Control	0.33 ± 0.15	0.35 ± 0.16	0.36 ± 0.17	0.193
IOP Right Eye (mmHg)	**Antioxidant**	15.8 ± 2.4	15.6 ± 2.2	15.5 ± 2.3	0.512
	Control	16.1 ± 2.6	16.0 ± 2.5	16.2 ± 2.7	0.624
IOP Left Eye (mmHg)	**Antioxidant**	15.5 ± 2.5	15.3 ± 2.4	15.2 ± 2.2	0.478
	Control	15.9 ± 2.3	16.1 ± 2.5	16.3 ± 2.6	0.417
RNFL Right Eye (µm)	**Antioxidant**	78.4 ± 6.8	78.1 ± 6.7	77.9 ± 6.6	0.214
	Control	79.1 ± 7.1	77.2 ± 6.9	75.4 ± 6.8	<0.001*
RNFL Left Eye (µm)	**Antioxidant**	77.9 ± 7.0	77.7 ± 6.8	77.5 ± 6.7	0.287
	Control	78.6 ± 6.5	76.8 ± 6.3	74.9 ± 6.2	<0.001*
GCC Right Eye (µm)	**Antioxidant**	74.6 ± 5.9	74.4 ± 5.8	74.2 ± 5.7	0.315
	Control	75.1 ± 6.2	73.6 ± 6.0	72.1 ± 5.8	<0.001*
GCC Left Eye (µm)	**Antioxidant**	74.2 ± 6.1	74.0 ± 6.0	73.8 ± 5.9	0.341
	Control	74.8 ± 5.8	73.1 ± 5.7	71.8 ± 5.6	<0.001*
MDA (nmol/mL)	**Antioxidant**	4.82 ± 0.72	4.15 ± 0.65	3.71 ± 0.61	<0.001*
	Control	4.75 ± 0.69	4.83 ± 0.73	4.96 ± 0.78	0.041*
TAC (mmol/L)	**Antioxidant**	1.21 ± 0.18	1.46 ± 0.20	1.68 ± 0.23	<0.001*
	Control	1.19 ± 0.17	1.17 ± 0.16	1.14 ± 0.18	0.083
SOD (U/mL)	**Antioxidant**	82.5 ± 9.4	91.7 ± 10.2	98.6 ± 11.1	<0.001*
	Control	83.1 ± 8.9	81.8 ± 9.2	80.6 ± 9.5	0.219
GPx (U/L)	**Antioxidant**	146.2 ± 18.5	161.8 ± 19.6	175.4 ± 21.2	<0.001*
	Control	145.8 ± 17.9	143.6 ± 18.2	141.9 ± 17.6	0.284

Correlation Between Oxidative Stress and Retinal Structure

Pearson correlation analysis was performed to assess the relationship between oxidative stress biomarkers and retinal structural parameters. A significant negative correlation was observed between MDA and RNFL thickness (r=-0.52, p=0.001), indicating that increased oxidative stress was associated with greater retinal nerve fiber layer loss. Positive correlations were found between TAC and RNFL thickness

(r=+0.46, p=0.003), SOD and GCC thickness (r=+0.49, p=0.002), and GPx and GCC thickness (r=+0.43, p=0.006), suggesting that improved antioxidant capacity and enzyme activity were associated with better preservation of retinal structures. Overall, the findings indicate that oxidative stress is linked with retinal neurodegenerative changes in POAG, while enhanced antioxidant status may contribute to protection of RNFL and GCC thickness.

Table 6. Pearson Correlation Analysis Between Oxidative Stress Biomarkers and Retinal Structural Parameters

Variables	r-value	p-value
MDA vs RNFL thickness	-0.52	0.001*
TAC vs RNFL thickness	+0.46	0.003*
SOD vs GCC thickness	+0.49	0.002*
GPx vs GCC thickness	+0.43	0.006*

DISCUSSION

The present randomized controlled trial demonstrated that oral antioxidant supplementation with CYT Softgel and Surbex Z, administered alongside standard glaucoma treatment, significantly reduced oxidative stress and preserved retinal structure in patients with primary open-angle glaucoma (POAG). Specifically, antioxidant supplementation was associated with significant reductions in malondialdehyde (MDA) levels and increases in total antioxidant capacity (TAC), superoxide dismutase (SOD), and glutathione peroxidase (GPx), accompanied by stabilization of retinal nerve fiber layer (RNFL) and ganglion cell complex (GCC) thickness over the 6-month follow-up period. In contrast, the control group exhibited progressive RNFL and GCC thinning despite stable intraocular pressure (IOP).

Our findings are consistent with those reported by Gherghel et al., who demonstrated significantly reduced systemic antioxidant defenses in patients with POAG, particularly lower glutathione levels, suggesting that impaired antioxidant capacity contributes to glaucomatous neurodegeneration. Similarly, Zanon-Moreno et al. found elevated malondialdehyde levels and decreased total antioxidant status in the aqueous humor of patients with POAG, supporting the hypothesis that oxidative stress is involved in glaucoma pathogenesis. [7, 9] The significant improvement in oxidative stress biomarkers observed in our study is also supported by recent evidence showing that

patients with POAG exhibit higher serum MDA levels and diminished antioxidant capacity compared with healthy individuals. Our findings extend this evidence by demonstrating that targeted antioxidant supplementation can modify these biomarkers favorably over time. Furthermore, the preservation of RNFL and GCC thickness in the antioxidant group aligns with emerging evidence suggesting that oxidative stress contributes directly to retinal ganglion cell apoptosis and structural damage. Previous studies have proposed that antioxidant-based neuroprotection may slow retinal neurodegeneration independent of IOP reduction, supporting the concept that glaucoma is a multifactorial neurodegenerative disease rather than solely an IOP mediated disorder. [22]

In contrast to some studies evaluating nutritional interventions in glaucoma, our study did not demonstrate significant reductions in IOP following antioxidant supplementation. This finding suggests that the beneficial effects of antioxidant therapy may occur through neuroprotective mechanisms rather than through alterations in aqueous humor dynamics or IOP regulation. Additionally, although some investigations have reported improvements in functional visual outcomes following nutritional supplementation, best-corrected visual acuity (BCVA) remained stable in both groups throughout our study. The absence of significant visual improvement may be explained by the relatively short follow-up duration, the slow progression of POAG, and the limited sensitivity of BCVA for detecting early glaucomatous changes. [23] Finally, while previous studies have primarily focused on cross-sectional associations between oxidative stress biomarkers and glaucoma severity, our study provides longitudinal evidence demonstrating significant correlations between changes in oxidative stress markers and retinal structural preservation. The observed negative correlation between MDA and RNFL thickness and the positive correlations between TAC, SOD, GPx, and GCC thickness suggest that enhanced antioxidant status may contribute to the preservation of retinal integrity. [22, 6]

The beneficial effects observed in this study may be attributed to the reduction of oxidative stress, attenuation of lipid peroxidation, and enhancement of endogenous antioxidant defenses, which collectively contribute to retinal ganglion cell protection and preservation of retinal structure. These findings support the potential role of antioxidant supplementation as an adjunct to conventional glaucoma treatment. However, larger multicenter randomized controlled trials with longer follow-up periods are

needed to confirm its long-term neuroprotective benefits and clinical applicability in patients with primary open-angle glaucoma.

Conclusion:

This randomized controlled trial demonstrates that antioxidant supplementation in patients with primary open-angle glaucoma was associated with significant improvement in oxidative stress status, reflected by reduced oxidative damage markers and increased antioxidant defense capacity. The preservation of retinal nerve fiber layer and ganglion cell complex thickness suggests a potential neuroprotective role of antioxidants by reducing retinal structural degeneration. Since no significant changes were observed in intraocular pressure or visual acuity, the observed benefits appear to be independent of conventional pressure-lowering effects and may occur through modulation of oxidative stress pathways.

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