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CLINICAL AND STRUCTURAL FEATURES OF COMORBID PRIMARY OPEN-ANGLE GLAUCOMA AND DRY AGE-RELATED MACULAR DEGENERATION

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Abstract

Objective. To evaluate structural and functional changes in patients with comorbid primary open-angle glaucoma (POAG) and dry age-related macular degeneration (AMD), compared to those with each condition in isolation, and to assess the impact of different therapeutic approaches including neuroprotective and antioxidant strategies.

Methods. This prospective, comparative-analytical clinical study included 168 patients (291 eyes) aged 60–82 years, divided into three groups: POAG only (n=46), dry AMD only (n=44), and comorbid POAG + AMD (n=78). All participants underwent standard ophthalmological assessment including visual acuity testing, intraocular pressure measurement, OCT imaging (macular zone, RNFL, GCL, choroidal thickness), OCT-angiography, and mfERG. Group 3 was subdivided based on therapy received: prostaglandins (3a), α2-agonist (brimonidine, 3b), and combined α2-agonist + AREDS 2 formula (3c). Observation lasted 12 months.

Results. Patients with comorbid POAG and AMD showed significantly worse BCVA, greater thinning of RNFL, GCL, macula, and choroid, as well as more severe functional impairment (lower MD and VFI) compared to isolated groups ($p<0,05$). Among therapeutic subgroups, combined treatment (3c) was associated with the least structural deterioration, highest visual field stability (86% of eyes),

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and lowest adverse event rate (7,1%), outperforming both monotherapy groups. **Conclusions.** Comorbidity of POAG and dry AMD leads to synergistic neurodegeneration and faster vision loss. OCT-derived structural parameters, especially choroidal and macular thinning, may serve as risk markers. Combination therapy with α2-agonists and antioxidants (AREDS 2) shows promise for preserving function in this high-risk group.

Keywords: Primary open-angle glaucoma, Age-related macular degeneration, Neurodegeneration, Optical coherence tomography, Antioxidant therapy.

Introduction

The aging of the global population has led to a steady increase in the prevalence of chronic degenerative eye diseases, particularly among individuals over 60 years of age. Among the most common causes of irreversible visual impairment in this age group are primary open-angle glaucoma (POAG) and age-related macular degeneration (AMD). Although these diseases differ in their pathogenesis, they share key features such as progressive neurodegeneration, retinal structural deterioration, and decline in visual function [1,2,3].

In clinical practice, cases of comorbid POAG and AMD are becoming increasingly common. However, this combined pathology poses significant diagnostic and therapeutic challenges. The simultaneous involvement of both central and peripheral parts of the visual system complicates differential diagnosis, interpretation of visual field testing and OCT data, and increases the risk of underdiagnosing disease progression. Additionally, the coexistence of macular and ganglion cell atrophy may represent a unique neurodegenerative phenotype associated with a more rapid decline in vision than in isolated POAG or AMD [4,5,6].

Despite the growing clinical relevance of this comorbidity, standardized diagnostic and treatment algorithms for patients with both POAG and AMD

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remain lacking. Identification of structural markers predictive of visual decline, such as macular and choroidal thinning, is essential for risk stratification and individualized patient monitoring. Furthermore, treatment selection in such cases is complex: while certain hypotensive agents may be appropriate for glaucoma alone, they may adversely affect choroidal circulation in the presence of AMD. Similarly, the role of neuroprotective agents and nutritional support remains understudied in comorbid cases [1,3,7,8].

Recent evidence [6,9,10] suggests that α2-agonists and antioxidant formulations such as AREDS 2 may offer protective benefits for both retinal ganglion cells and the macular region. However, no comprehensive prospective studies have compared structural and functional changes, or therapeutic efficacy, in patients with isolated versus comorbid POAG and dry AMD using modern imaging techniques such as OCT, OCT-angiography, and multifocal ERG.

Objective of the Study

To perform a comprehensive evaluation of clinical and functional features, diagnostic risk markers, and treatment efficacy in patients with comorbid primary open-angle glaucoma and dry age-related macular degeneration, compared to those with each condition in isolation.

Material and Methods

A prospective, comparative-analytical clinical study with elements of longitudinal follow-up was conducted as part of this investigation. The total duration of observation was 12 months, with the possibility of extension to 18 months depending on the severity and progression rate of morphofunctional changes. The aim of the study was to perform a comprehensive assessment of the clinical and functional characteristics of isolated and comorbid POAG and dry AMD, with a particular focus on identifying structural risk markers for visual deterioration and analyzing therapeutic approaches.

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A total of 168 patients (291 eyes) aged 60 to 82 years who met the inclusion criteria were enrolled. The mean age of participants was 69,7±5,4 years. Females predominated in the sample - 98 patients (58,3%) compared to 70 males (41,7%). All patients underwent a complete ophthalmological examination prior to inclusion and signed informed consent for participation. Group assignment was based on a confirmed diagnosis of isolated or comorbid POAG and/or dry AMD. According to the study design, all participants were divided into three main groups: Group 1 included 46 patients (88 eyes) with isolated POAG; Group 2 consisted of 44 patients (76 eyes) with isolated dry AMD; and Group 3 included 78 patients (127 eyes) with comorbid POAG and dry AMD. To assess the impact of different treatment regimens, patients in Group 3 were further divided into three subgroups: Subgroup 3a (n=26) received standard hypotensive therapy with prostaglandin analogs; Subgroup 3b (n=24) received neuroprotective therapy with the α2-agonist brimonidine; and Subgroup 3c (n=28) received combination therapy consisting of brimonidine plus nutritional antioxidant support based on the AREDS 2 formula.

Inclusion criteria were: age over 60 years; a confirmed diagnosis of POAG and/or dry AMD; the ability to undergo a complete ophthalmological evaluation; and signed informed consent. Exclusion criteria included: wet AMD, secondary or angle-closure glaucoma, other ocular diseases affecting the macula or optic nerve, history of intraocular surgery within 6 months prior to enrollment, severe systemic or neurological illness, or inability to comply with visit schedules.

All participants underwent a standardized ophthalmologic examination, including visual acuity testing, intraocular pressure measurement using Goldmann applanation tonometry, static automated perimetry (Humphrey Field Analyzer 24-2), and optical coherence tomography (OCT) to assess the macular region, RNFL, GCL, and subfoveal choroidal thickness. OCT-angiography was performed to evaluate macular and peripapillary blood flow, and multifocal

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electroretinography (mfERG) was used to assess the functional state of the central retina.

Statistical analysis was performed using SPSS v.25.0 and GraphPad Prism v.9.0. Methods included descriptive statistics, Student's t-test, one-way ANOVA, correlation analysis, and multiple regression modeling. A p-value of <0,05 was considered statistically significant for all tests.

Results

The study included a total of 168 patients (291 eyes) distributed across three groups based on diagnosis. Group 1 consisted of 46 patients with isolated POAG, Group 2 included 44 patients with isolated dry AMD, and Group 3 included 78 patients with comorbid POAG and dry AMD. The mean age of participants was 69,7±5,4 years, with a female predominance. There were no statistically significant differences in age or sex distribution across groups (p>0,05) (Table 1). A comparative analysis of baseline visual and structural parameters revealed significant intergroup differences. Patients in the comorbid group (Group 3) demonstrated the lowest best-corrected visual acuity (BCVA) compared to POAG and dry AMD groups (p<0,05). Mean intraocular pressure (IOP) was elevated in both POAG and comorbid groups relative to the AMD group (p<0,05).

Table 1. Baseline Demographic and Clinical Characteristics of the Study Groups.

Parameter	Group 1 (POAG) (n=46)	Group 2 (Dry AMD) (n=44)	Group 3 (POAG + Dry AMD) (n=78)	p
Number of eyes	88	76	127	—
Mean age (years)	69,2±5,6	70,1±5,1	69,9±5,4	0,47
Female, n (%)	25 (54,3%)	28 (63,6%)	45 (57,7%)	0,62
Male, n (%)	21 (45,7%)	16 (36,4%)	33 (42,3%)	
Mean IOP (mmHg)	22,6±2,1	15,8±1,9	21,9±2,4	<0,001
Best-corrected visual acuity (BCVA)	0,72±0,12	0,58±0,10	0,49±0,14	<0,001

P-values were calculated using ANOVA or chi-square test where appropriate.

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Assessment of optical coherence tomography (OCT) and visual field parameters (Table 2) showed significantly more pronounced thinning of retinal nerve fiber layer (RNFL) and ganglion cell layer (GCL) in the comorbid group, indicating more advanced neurodegenerative changes. Additionally, macular and subfoveal choroidal thickness were significantly reduced in patients with comorbid POAG and AMD ($p<0,05$), suggesting synergistic structural atrophy. Functional testing showed the worst mean deviation (MD) and lowest Visual Field Index (VFI) values in the comorbid group, supporting the hypothesis of accelerated functional decline in combined pathology.

Table 2. Structural and Functional Parameters According to OCT and Visual Field Testing.

Parameter	POAG (n=88 eyes)	Dry AMD (n=76 eyes)	POAG + Dry AMD (n=127 eyes)	*p*
RNFL thickness (µm)	84,1±9,8	97,2±7,4	79,6±10,5	*<0,05*
GCL thickness (µm)	75,3±7,6	82,9±6,2	70,5±8,1	*<0,05*
Central macular thickness (µm)	262,4±15,5	248,1±14,8	238,9±17,3	*<0,05*
Subfoveal choroidal thickness (µm)	203,6±24,4	187,3±20,6	170,2±22,1	*<0,05*
Mean deviation (MD), dB	−3,1±1,8	−2,2±1,6	−5,4±2,3	*<0,05*
Visual Field Index (VFI), %	89,4±6,7	92,1±5,5	78,6±8,9	*<0,05*

RNFL – retinal nerve fiber layer; GCL – ganglion cell layer; MD – mean deviation; VFI – Visual Field Index.

Further stratification within Group 3 based on treatment modality revealed distinct differences in disease progression and therapeutic outcomes (Table 3). Patients receiving α2-agonist monotherapy (Group 3b) and combined α2-agonist plus AREDS 2 nutritional support (Group 3c) demonstrated less GCL and macular thinning over 12 months compared to those on prostaglandin-based therapy (Group 3a) ($p < 0,05$). The greatest stabilization of visual field function (VFI) was observed in the combination therapy group (Group 3c), with 86% of

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eyes maintaining stable VFI (<5% change), compared to 63% in the prostaglandin group (p=0,01). Incidence of adverse events was also lower in the combination group.

Table 3. Therapeutic Outcomes in Patients with Comorbid POAG and Dry AMD (Group 3).

Parameter	3a: Prostaglandins (n=26)	3b: α2-agonist (n=24)	3c: α2-agonist + AREDS 2 (n=28)	*p*
IOP reduction (mmHg)	−3,1±1,2	−4,2±1,3	−4,4±1,1	*0,002*
Change in GCL thickness at 12 months (µm)	−5,6±1,9	−3,2±1,5	−2,4±1,6	*<0,05*
Change in macular thickness at 12 months (µm)	−12,3±3,4	−7,8±3,1	−5,1±2,8	*<0,05*
MD progression at 12 months (dB)	−1,6±0,7	−0,9±0,6	−0,6±0,5	*<0,05*
VFI stabilization (%)	63%	79%	86%	*0,01*
Adverse events, n (%)	7 (26,9%)	3 (12,5%)	2 (7,1%)	—

Discussion

This study provides a comprehensive comparison of structural and functional changes in patients with isolated POAG, AMD, and their comorbid presentation. The results demonstrate that patients with coexisting POAG and dry AMD exhibit a more pronounced and accelerated deterioration in both retinal structure and visual function, confirming the hypothesis of a synergistic neurodegenerative effect in comorbid ophthalmic pathology.

The significant thinning of the RNFL and GCL observed in the comorbid group compared to the isolated disease groups suggests that glaucomatous damage may be amplified in the presence of macular atrophy, and vice versa. These findings are consistent with previous research showing that ganglion cell degeneration is more pronounced in AMD patients than previously assumed, especially in the context of combined disease processes [1,5,9].

Moreover, the greater reduction in macular thickness and subfoveal choroidal thickness in the comorbid group underscores the compounded structural burden

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associated with simultaneous retinal and optic nerve degeneration. As the choroid plays a crucial role in macular nourishment and metabolic support, its thinning may exacerbate photoreceptor and ganglion cell loss, leading to accelerated visual decline. These findings support the need to include choroidal thickness as a risk stratification marker during OCT-based follow-up [2,6].

Functionally, the lowest MD and VFI values were recorded in the comorbid group, further indicating that visual field loss progresses more aggressively when both diseases are present. This underlines the need for more frequent and integrated visual field and OCT monitoring in these patients [3,7].

Importantly, the therapeutic component of this study revealed clinically relevant differences. Patients treated with α2-agonists (brimonidine) and those receiving combined therapy (α2-agonist + AREDS 2) showed significantly slower progression of GCL and macular thinning over 12 months. The combined therapy group also had the highest percentage of VFI stabilization (86%) and the lowest rate of adverse events, highlighting the potential neuroprotective and vasoprotective benefits of this approach. In contrast, patients on prostaglandin analogs experienced greater structural loss, which may be related to their known effect of reducing peripapillary and choroidal perfusion, potentially worsening macular atrophy in AMD patients.

Taken together, these findings suggest that patients with comorbid POAG and AMD represent a distinct clinical subgroup that requires modified diagnostic and therapeutic strategies. The traditional monotherapy approach for POAG may be insufficient or even harmful in the context of coexisting macular degeneration. Instead, therapies that combine intraocular pressure control, neuroprotection, and antioxidant support may provide superior outcomes.

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