ORIGINAL ARTICLE

CONTENTS

Factors affecting the progression of glaucomatous optic neuropathy after antiglaucoma surgery in patients with primary open-angle glaucoma

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ABSTRACT

Aim: To identify and analyze the key clinical, structural, and functional factors influencing the progression of glaucomatous optic neuropathy after antiglaucoma surgical intervention in patients with POAG, as well as to determine their prognostic value for optimizing postoperative patient management.

Materials and Methods: A retrospective observational cohort study included 56 patients (56 operated eyes) with primary open-angle glaucoma who underwent unilateral antiglaucoma surgery and were followed dynamically in the postoperative period. Based on follow-up results, patients were divided into two groups: with postoperative disease progression (n = 20) and without progression (n = 36).

Results: Postoperative disease progression was not associated with age, sex, axial length, or phacomorphic syndrome. Besides, disease progression was significantly associated with elevated plasma LOX1 concentration (≥ 80.1 pg/mL), reduced neuroretinal rim area (≤ 0.7 mm^2), and elevated preoperative IOP (≥ 25 mmHg). In multivariate logistic regression analysis, reduced neuroretinal rim area ≤ 0.7 mm^2 (OR = 12.9; 95% CI: 3.4–49.1; $p < 0.05$) and preoperative IOP ≥ 25 mmHg (OR = 7.6; 95% CI: 1.9–29.4; $p < 0.05$) remained independent predictors of postoperative disease progression, while plasma LOX1 showed moderate discriminative performance (AUC = 0.664).

Conclusions: Postoperative progression of glaucomatous optic neuropathy in patients with primary open-angle glaucoma is determined by a combination of structural, biomechanical, and systemic vascular-related factors. Preoperative IOP, neuroretinal rim morphology, and plasma LOX1 levels have prognostic significance and should be considered for postoperative risk stratification and individualized follow-up.

KEY WORDS: glaucoma, surgery, inflammation, optometry, diagnostics, trabecula

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INTRODUCTION

Glaucoma remains one of the leading causes of irreversible blindness worldwide. According to global epidemiological projections, the number of patients with glaucoma may reach 112 million by 2040 [1], highlighting the urgent need for early diagnosis and effective control of disease progression. Primary open-angle glaucoma (POAG) is the most common type of glaucoma and is characterised by a chronic, slowly progressive, and often asymptomatic course, which frequently leads patients to seek medical attention at advanced stages when visual loss becomes irreversible [1, 2].

Despite the variety of pathogenetic mechanisms involved, elevated intraocular pressure (IOP) remains the only reliably established modifiable risk factor for the progression of glaucomatous optic neuropathy. International randomised clinical trials have confirmed that reducing IOP slows both visual field deterioration and structural changes of the optic nerve head in patients with glaucoma across different stages [2]. Nevertheless, postoperative IOP stability is equally important, as it has been shown that IOP fluctuations, even when the mean pressure remains within normal limits, can accelerate glaucoma progression. The postoperative period in POAG patients is considered particularly critical because, even after achieving target IOP, some patients continue to demonstrate structural or functional deterioration. It is known that after surgical intervention, structural changes of the optic nerve head and retina may continue to evolve despite satisfactory IOP levels, which may be partially explained by vascular, biomechanical, and neurodegenerative influences [2, 3].

Special attention in the pathogenesis of glaucoma progression is given to systemic biological mechanisms, including oxidative stress, endothelial dysfunction, inflammatory responses, and reduced neurotrophic sup-

port, all of which can affect both the resistance of ocular tissues to increased IOP and their ability to recover after surgery [4, 5]. In this context, the investigation of plasma biomarkers such as lysyl oxidase-1 (LOX1)—a molecule involved in oxidative tissue damage and apoptosis—has gained particular importance [5]. Elevated LOX1 levels may be associated with increased susceptibility of the optic nerve to damage, making this biomarker a promising candidate for assessing the risk of glaucoma progression, including in the postoperative period.

Given the complexity of POAG pathogenesis and the variety of factors influencing postoperative outcomes, identifying clinical and biochemical predictors of progression is essential for optimising postoperative management [1, 2, 6]. The identification of these predictors not only helps detect high-risk patients but also contributes to developing personalised monitoring and treatment strategies tailored to the individual risk profile. Therefore, our study aimed to provide a comprehensive assessment of clinical, morphological, and biochemical factors potentially influencing the progression of glaucomatous optic neuropathy after antiglaucoma surgery in patients with POAG.

AIM

The aim of the study was to identify and analyze the key clinical, structural, and functional factors influencing the progression of glaucomatous optic neuropathy after antiglaucoma surgical intervention in patients with POAG, as well as to determine their prognostic value for optimizing postoperative patient management.

MATERIALS AND METHODS

A retrospective observational cohort study was conducted to evaluate factors associated with the progression of glaucomatous optic neuropathy after antiglaucoma surgery in patients with POAG. All patients were subjected to dynamic postoperative follow-up, during which clinical, structural, and functional parameters were assessed in the postoperative period in order to determine the presence or absence of glaucoma progression. The study design included comparison of preoperative baseline data with follow-up observations obtained after surgical intervention. The surgical procedure performed was a minimally invasive tunnel trabeculopuncture. The mean duration of postoperative follow-up was 43 months. The main study group included 56 patients (56 operated eyes) with primary open-angle glaucoma who underwent antiglaucoma surgical intervention. Only patients with unilateral surgery were included in the analysis; therefore, one operated eye per patient was evaluated, and the unit of analysis was the individual patient.

The study group consisted of 26 men (46.4%) and 30 women (53.6%), with a median (Me) age of 63.0 years (interquartile range [IQR]: 56.0–71.0). According to the results of dynamic postoperative follow-up, disease progression was detected in 20 patients (35.7%), while 36 patients (64.3%) demonstrated no signs of progression. Based on these findings, patients were divided into two groups for further analysis: a group without postoperative glaucoma progression and a group with postoperative glaucoma progression.

All patients enrolled in the study underwent comprehensive ophthalmic examinations and treatment at the clinical facilities of the Department of Ophthalmology and Optometry of Postgraduate Education, Institute of Postgraduate Education, Bogomolets National Medical University.

All patients underwent a comprehensive ophthalmic evaluation, which, in addition to standard examinations, included the assessment of horizontal cup-to-disc ratio; vertical cup-to-disc ratio; presence of β-zone peripapillary atrophy; presence of neuroretinal rim notching in the inferior sector; and focal loss of the retinal nerve fibre layer (RNFL).

Quantitative measurements were obtained for the ganglion cell complex in the peripapillary and paramacular regions. The temporal peripapillary RNFL thickness was assessed, and the volume of the neuroretinal rim was measured, alongside its correlation with the qualitative characteristics of the corresponding optic nerve head regions.

Fig. 1 presents fundus photography and optical coherence tomography (OCT) images of the optic nerve head (ONH) of patient H., 65 years old, diagnosed with stage III POAG in the right eye and stage II POAG in the left eye.

Measurement of the LOX1 biomarker level in plasma was performed using a solid-phase enzyme-linked immunosorbent assay (ELISA). Blood plasma samples were collected in sterile Eppendorf tubes in the clinical diagnostic laboratory as residual material obtained after routine blood testing. Plasma was prepared according to standard protocols via centrifugation, then frozen at −20°C and transported to the research laboratory under controlled cold-chain conditions. LOX1 concentrations were measured using the Human LOX-1 ELISA Kit (Invitrogen, Thermo Scientific, USA; Catalogue No. EHOLR1; Lot 371031324; sensitivity 2 pg/mL), following the manufacturer's instructions.

Statistical analysis was performed using the EZR software package (version 1.55; R statistical environment). The distribution of continuous variables was assessed

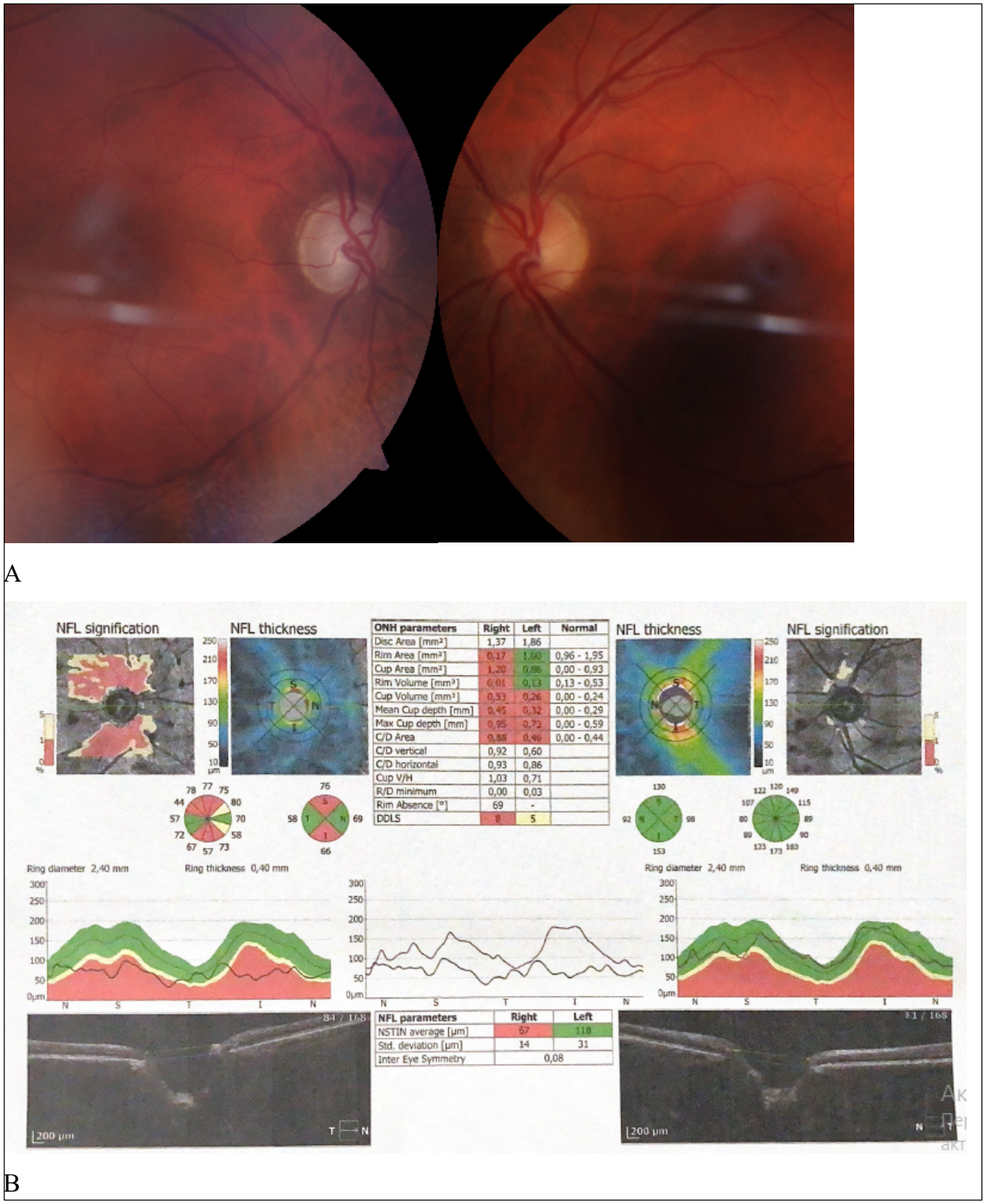

Fig. 1. Patient H., 65 years old, POAG stage III (right eye), stage II (left eye): A – fundus photo of the ONH; B – OCT results
Picture taken by the authors

using the Shapiro–Wilk test. As most continuous variables did not follow a normal distribution, quantitative data were presented as Me (IQR) and analysed using non-parametric statistical methods. Comparisons between two independent groups were performed using the Mann–Whitney U test. Categorical variables were

Table 1. Characteristics of patients with and without postoperative disease progression

Variable	Disease progression (n =20)	Without disease progression (n =36)	p
Age, years	64.5 (56.0-72.0)	63.0 (57.0-71.0)	0.751
Women (N = 30)	10 (50.0 %)	20 (55.6 %)	0,783
Men (N = 26)	10 (50.0 %)	16 (44.4 %)	
Phacomorphic syndrome (N = 23)	11 (55.0 %)	14 (38.9 %)	0,275
Without phacomorphic syndrome (N = 33)	9 (45.0 %)	22 (61.1 %)	
Anterior-posterior axis, mm	24.54 (23.18–24.98)	23.56 (22.83–24.91)	0.235
Neuroretinal area, mm^2	0.75 (0.70–0.80)	0.50 (0.445–0.50)	< 0.001
IOP, mm Hg	28.5 (25.5–31.0)	19.0 (18.0–23.0)	0.001
LOX1, pg/mL	49.65 (33.42–170.79)	41.40 (29.68–56.70)	0.045

Source: compiled by the authors of this study

expressed as absolute values and percentages and compared using Fisher's exact test.

Postoperative glaucoma progression (presence or absence of progression) was treated as a binary dependent variable for regression modelling. Univariate analysis was initially performed to assess the association between individual clinical, anatomical, and biochemical variables and postoperative progression of glaucomatous optic neuropathy. Variables demonstrating a potential association with postoperative progression in univariate analysis were subsequently included in a logistic regression model. Multivariate logistic regression analysis was applied to identify independent predictors of postoperative glaucomatous optic neuropathy progression. The results of logistic regression analysis were expressed as odds ratios (ORs) with corresponding 95% confidence intervals (CIs). Receiver operating characteristic (ROC) analysis was performed for variables identified as significant predictors in logistic regression analysis to evaluate their discriminative ability. The area under the ROC curve (AUC), sensitivity, and specificity were calculated, and optimal cut-off values were determined based on ROC curve analysis. A p-value < 0.05 was considered statistically significant.

ETHICS

The study was conducted in accordance with the main provisions of the Council of Europe Convention on Human Rights and Biomedicine, the Declaration of Helsinki of the World Medical Association (1964, with subsequent amendments including the 2000 version), as well as current regulatory acts of Ukraine: "Fundamentals of Ukrainian Legislation on Health Care" (1993), "On the Protection of Personal Data" (2010), and "On Higher Education" (2017). All patients were fully informed about the nature of the study and provided written informed consent for diagnostic examination and treatment.

FRAMEWORK

The work was carried out at the Department of Ophthalmology and Optometry of Postgraduate Education of the Institute of Postgraduate Education of Bogomolets National Medical University and is a fragment of the scientific research project «Theoretical and practical aspects of improving clinical and experimental methods of diagnosis, treatment and prevention of diseases and injuries of the organ of vision and their complications» (state registration number 0123U104207; term: 2023-2026).

RESULTS

All ophthalmological parameters presented in the following tables were assessed in the operated eyes only. As only patients who underwent unilateral antiglaucoma surgery were included in the study, one operated eye per patient was analysed. Based on dynamic postoperative follow-up, patients were divided into two groups: those with postoperative glaucomatous optic neuropathy progression (n = 20) and those without disease progression (n = 36) (Table 1).

The median age of the entire study population was 63.0 years (IQR: 56.0–71.0). There was no statistically significant difference in age between patients with postoperative disease progression and those without progression (median 64.5 years [IQR: 56.0–72.0] vs 63.0 years [IQR: 57.0–71.0]; p = 0.751).

Sex distribution did not differ significantly between groups. Among women (n = 30), postoperative progression was observed in 10 patients (50.0%), while 20 patients (55.6%) showed no progression. Among men (n = 26), progression occurred in 10 cases (50.0%), and 16 patients (44.4%) had no progression (p = 0.783).

Phacomorphic syndrome was not significantly associated with postoperative disease progression.

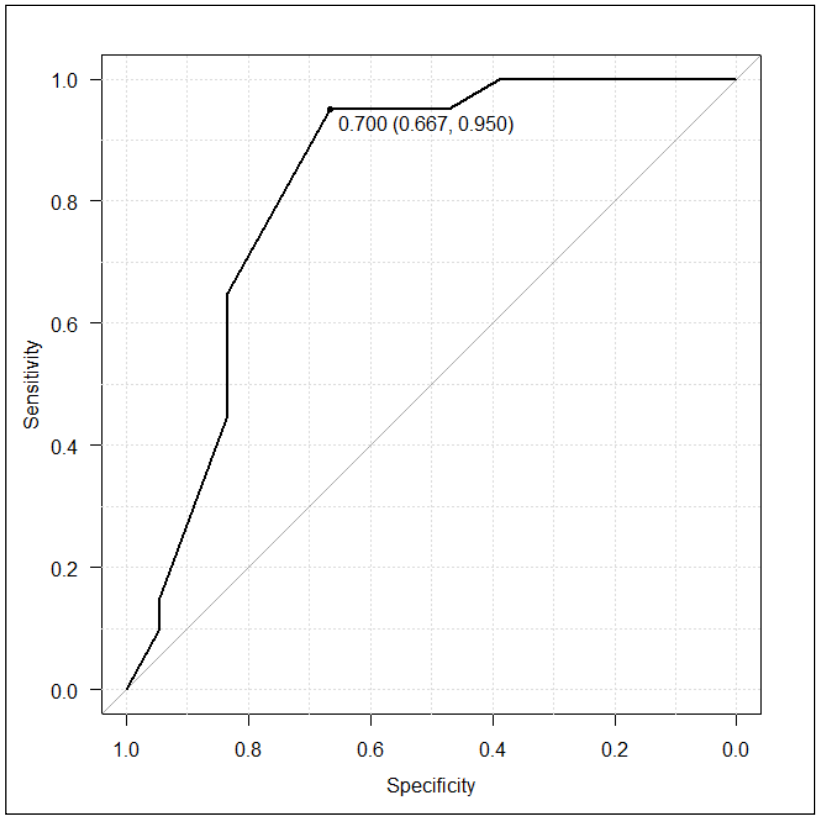

Fig. 2. ROC curve of the logistic model predicting the frequency of POAG progression after surgical antiglaucoma intervention depending on the size of the neuroretinal strip area before surgery. AUC = 0.824
Picture taken by the authors

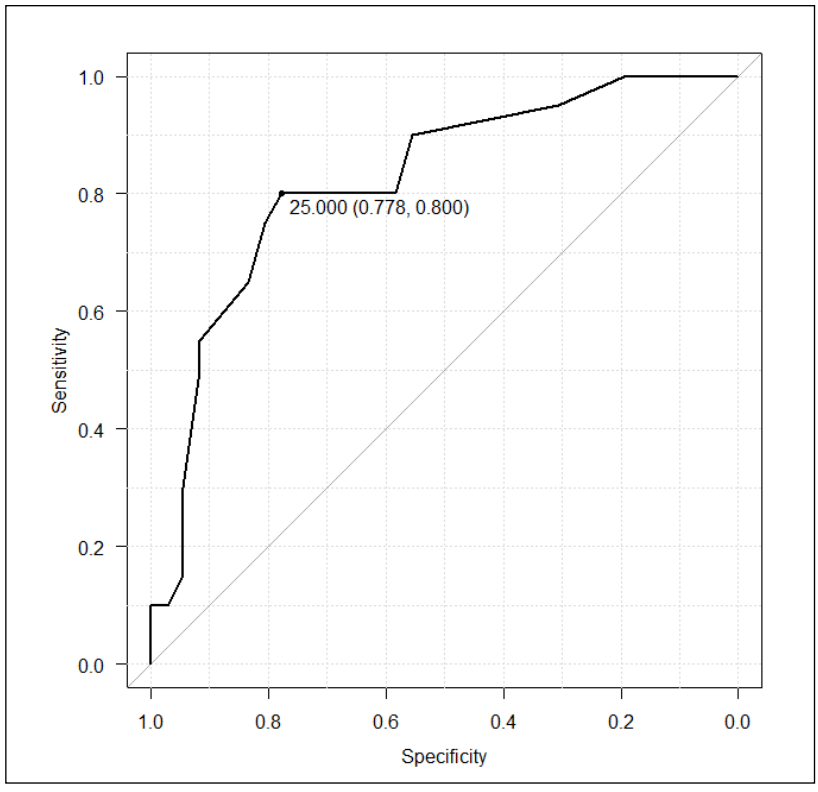

Fig. 3. ROC curve of the logistic model predicting the frequency of POAG progression after surgical antiglaucoma intervention depending on the value of IOP before surgery. AUC = 0.822
Picture taken by the authors

Progression was detected in 11 of 23 patients (55.0%) with phacomorphic syndrome and in 9 of 33 patients (45.0%) without phacomorphic syndrome (p = 0.275).

Preoperative intraocular pressure differed significantly between the two groups. Patients with postoperative disease progression had a substantially higher IOP compared with those without progression (median 28.5 mmHg [IQR: 25.5–31.0] vs 19.0 mmHg [IQR: 18.0–23.0], Mann–Whitney U test, p < 0.001).

Neuroretinal rim area was significantly lower in patients with postoperative disease progression than in patients without progression. The median

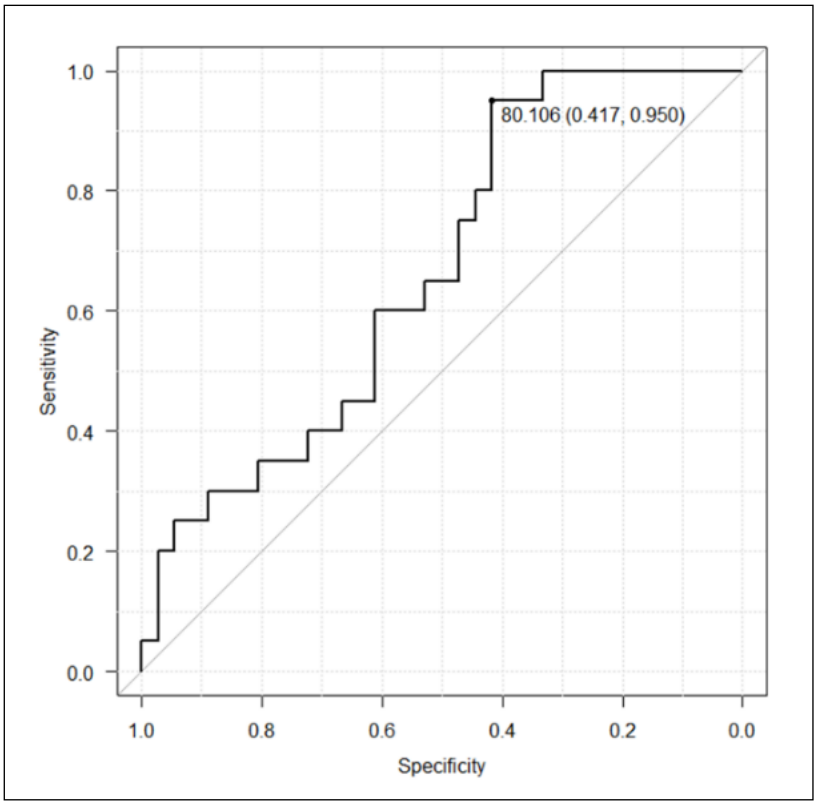

Fig. 4. ROC curve of the logistic model predicting the frequency of POAG progression after antiglaucoma surgery depending on the value of LOX1 in the blood plasma of glaucoma patients before surgery. AUC = 0.664
Picture taken by the authors

Table 2. The certain binary risk factors in the groups of patients with and without postoperative disease progression

Variable	Disease progression (n =20)	Without disease progression (n =36)	p
Neuroretinal area ≤ 0,7 mm² (N = 23)	18 (90.0 %)	6 (16.7 %)	<0.001
Neuroretinal area > 0,7 mm² (N = 33)	2 (10.0 %)	30 (83.3 %)	
IOP < 25 mmHg (N = 20)	2 (10.0 %)	18 (50.0 %)	0.003
IOP ≥ 25 mmHg (N = 36)	18 (90.0 %)	18 (50.0 %)	
LOX1 ≥ 80.1 pg/mL (N = 19)	17 (85.0 %)	2 (5.6 %)	<0.001
LOX1 < 80.1 pg/mL (N = 37)	3 (15.0 %)	34 (94.4 %)	

Source: compiled by the authors of this study

Table 3. Multivariate analysis of factors associated with postoperative glaucomatous optic neuropathy progression

Variable	OR	95% CI	p
Neuroretinal rim area ≤ 0.7 mm²	12.9	3.4 – 49.1	< 0.05
Preoperative intraocular pressure ≥ 25 mmHg	7.6	1.9 – 29.4	< 0.05
LOX1 ≥ 80.1 pg/mL	2.2	0.8 – 6.1	0.12

Source: compiled by the authors of this study

neuroretinal rim area in the progression group was 0.50 mm² (IQR: 0.445–0.50), compared with 0.75 mm² (IQR: 0.70–0.80) in the non-progression group ($p < 0.001$).

Plasma LOX1 concentrations showed a statistically significant difference between groups. Patients without postoperative disease progression had lower median LOX1 levels compared with patients with progression (median 41.40 pg/mL [IQR: 29.68–56.70] vs 49.65 pg/mL [IQR: 33.42–170.79]; $p = 0.045$) (Table 1).

The ROC analysis was performed for variables that demonstrated a statistically significant association with postoperative glaucomatous optic neuropathy progression in order to assess their individual discriminative ability. It should be emphasised that ROC analysis represents a single-factor assessment and does not account for interactions between variables within the multivariate model.

ROC analysis demonstrated a high discriminative ability of the preoperative neuroretinal rim area for

predicting postoperative disease progression. A cut-off value of < 0.7 mm^2 was associated with a high risk of progression, yielding an AUC of 0.824 (95% CI: 0.716–0.932). Sensitivity and specificity at this threshold were 95.0% and 66.7%, respectively (Fig. 2).

Preoperative IOP also showed high discriminative performance in ROC analysis. An IOP threshold of ≥ 25 mmHg was associated with postoperative progression, with an AUC of 0.822 (95% CI: 0.706–0.937). At this cut-off value, sensitivity was 80.0%, and specificity was 77.8% (Fig. 3).

Plasma LOX1 concentration demonstrated moderate discriminative ability for postoperative progression. A preoperative LOX1 cut-off value of ≥ 80.1 pg/mL was associated with postoperative progression, with an AUC of 0.664 (95% CI: 0.520–0.808). Sensitivity and specificity at this threshold were 95.0% and 41.7%, respectively (Fig. 4).

Based on the results of ROC analysis, optimal cut-off values were determined for continuous variables demonstrating significant discriminative ability. These cut-off points were subsequently used to categorise patients and to perform a comparison of categorical (binary) risk factors between patients with and without postoperative disease progression (Table 2).

According to the data in Table 2, all the studied binary variables demonstrated a statistically significant association with postoperative glaucomatous optic neuropathy progression. In the next step, the neuroretinal area, IOP, and LOX1, adjusted for other previously described parameters, were evaluated using multivariate logistic regression analysis.

As shown in Table 3, multivariate logistic regression analysis identified reduced neuroretinal rim area (< 0.7 mm^2) and elevated preoperative IOP (> 25 mmHg) as independent predictors of postoperative disease progression. A neuroretinal rim area < 0.7 mm^2 was associated with a markedly increased risk of progression (OR = 12.9, 95% CI: 3.4–49.1; $p < 0.001$), while preoperative IOP > 25 mmHg also remained an independent predictor (OR = 7.6, 95% CI: 1.9–29.4; $p < 0.001$).

Plasma LOX1 concentration, although significantly associated with disease progression in univariate analysis, did not retain independent predictive significance in the multivariate logistic regression model after adjustment for structural and pressure-related factors (OR = 2.2, 95% CI: 0.8–6.1; $p = 0.12$) (Table 3).

DISCUSSION

The findings of this study indicate that, following antiglaucoma surgery in patients with primary open-angle glaucoma, the progression of glaucomatous optic neuropathy is determined not only by the achieved postoperative IOP level but also by a combination of preoperative morphofunctional characteristics and, likely, vascular factors. This aligns with the modern understanding of glaucoma as a multifactorial disease in which structural and functional deterioration may persist even after successful surgical reduction of IOP. Several studies have demonstrated that after trabeculectomy, some patients experience a slowing of visual field loss, whereas others continue to show progression despite satisfactory IOP control [7–9].

Our analysis confirms that high preoperative IOP is an important predictor of future glaucomatous optic neuropathy progression, even after successful surgical treatment. This observation is consistent with published findings showing that eyes with higher preoperative IOP peaks exhibit worse preservation of macular ganglion cell complex (GCC) parameters in subsequent years. Specifically, eyes with lower preoperative peak IOP demonstrate better preservation of the thickness of the macular nerve fiber layer (mNFL), macular ganglion cell layer (mGCL), and inner plexiform layer (mIPL) compared to eyes with higher peak IOP, even when their postoperative IOP levels are similar [8, 10].

Another significant factor is the status of the neuroretinal rim. A thinner neuroretinal rim and a larger cup-to-disc ratio can be considered morphological indicators of increased vulnerability of the optic nerve head to subsequent damage. Although partial reversal of cupping and changes in optic nerve head topography have been observed in some patients after trabeculectomy, these morphometric improvements do not always correspond to long-term structural stability [8]. Therefore, the baseline configuration of the optic nerve head — including neuroretinal rim condition — should be considered a prognostically significant parameter during postoperative follow-up.

The results of previous studies indicate that achieving target IOP alone may not fully prevent disease progression. One plausible explanation is the influence of vascular mechanisms [9]. For example, a more rapid decrease in peripapillary vessel density following trabeculectomy has been associated with visual field deterioration, suggesting an independent contribution of microcirculatory impairment. Epidemiological and clinical data also indicate the role of systemic vascular dysregulation — including hypotension, blood pressure variability, and impaired autoregulation — in maintaining the risk of progression after surgery [10].

Although direct clinical evidence regarding the role of LOX1 in the progression of glaucoma after antiglaucoma surgery is still limited, known mechanisms of its biological activity support its potential as an important factor in the pathogenesis of glaucomatous optic neuropathy. LOX1 is a transmembrane receptor whose expression increases

in response to oxidative stress, dyslipidaemia, endothelial dysfunction, and tissue ischaemia — all of which are processes associated with vascular forms of glaucoma [5].

Retinal ganglion cells (RGCs) are especially sensitive to microcirculatory disturbance and oxidative stress. Activation of LOX1 triggers the generation of reactive oxygen species (ROS), reduced nitric oxide (NO) bioavailability, and initiation of apoptosis through caspase-dependent pathways [5, 10].

In glaucoma, characterised by reduced perfusion pressure and impaired autoregulation, LOX1 activation may exacerbate the metabolic vulnerability of RGCs, thereby increasing the risk of progression even when postoperative IOP remains within the target range [4].

Thus, even after achieving satisfactory postoperative IOP control, elevated systemic LOX1 levels may contribute to ongoing microvascular ischaemia and RGC loss, clinically manifesting as continued progression [5, 7].

The limitations of this study include the relatively small sample size and its retrospective design. Furthermore, because glaucoma is inherently multifactorial, the dominant influence of preoperative IOP, optic nerve head morphology, and vascular factors must be interpreted with caution [4, 9].

Nevertheless, LOX1 appears to be a promising pathogenic biomarker contributing to progression risk, although it should not be considered a sole determinant. Patients with elevated LOX1 levels should be regarded as high-risk and require closer ophthalmic follow-up and more intensive postoperative monitoring [5–7].

Overall, the combination of elevated preoperative IOP, morphological parameters, and systemic biochemical markers may enable more accurate risk stratification and support the development of personalised postoperative management strategies for patients with primary open-angle glaucoma.

CONCLUSIONS

1. Postoperative progression of glaucomatous optic neuropathy in patients with POAG is a multifactorial process that may occur despite successful surgical reduction of intraocular pressure. This underscores the importance of identifying patients at increased risk during postoperative follow-up.
2. Elevated preoperative IOP ($\geq$ 25 mmHg) was identified as an independent predictor of postoperative disease progression. Patients with higher preoperative IOP demonstrated a significantly increased risk of structural and functional deterioration after surgery.
3. Reduced neuroretinal rim area ($\leq$ 0.7 mm^2) was found to be an independent predictor of postoperative glaucomatous optic neuropathy progression. This parameter reflects advanced baseline optic nerve damage and is associated with a markedly unfavorable postoperative course.
4. Plasma LOX1 concentration showed a significant association with disease progression in univariate analysis; however, it did not retain independent predictive value after adjustment for structural and pressure-related factors. These findings suggest that LOX1 may serve as an adjunctive biomarker, although its clinical relevance requires further confirmation in larger prospective studies.

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CONFLICT OF INTEREST
The Authors declare no conflict of interest

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