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GENETIC ARCHITECTURE AND MOLECULAR BIOMARKERS OF PRIMARY OPEN-ANGLE GLAUCOMA: IMPLICATIONS FOR RISK STRATIFICATION AND PERSONALIZED THERAPY

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ABSTRACT

Introduction. Primary open-angle glaucoma (POAG) is a genetically heterogeneous optic neuropathy in which elevated intraocular pressure, retinal ganglion cell susceptibility, vascular regulation, extracellular-matrix remodeling, mitochondrial dysfunction, and immune signaling interact. Aim. To synthesize current evidence on monogenic variants, common polygenic susceptibility, molecular biomarkers, and emerging genotype-informed therapeutic strategies in POAG. Materials and methods. A narrative review was conducted using PubMed/MEDLINE-indexed publications identified with combinations of the terms "primary open-angle glaucoma," "genetics," "genome-wide association study," "polygenic risk score," "biomarker," "proteomics," "microRNA," and "gene therapy." Priority was given to multi-ancestry genetic studies, systematic reviews, longitudinal cohorts, and experimentally validated translational studies published through July 2026. Results. Rare variants in MYOC, OPTN, and TBK1 explain a small but clinically important fraction of familial or early-onset disease. Genome-wide analyses have identified hundreds of loci that converge on aqueous-outflow regulation, optic nerve development, vascular biology, extracellular matrix, and retinal ganglion cell survival. Polygenic risk scores are associated with disease onset, progression, and earlier need for surgery, but portability across ancestries remains limited. Candidate biomarkers in aqueous humor, tears, and blood include cytokines, complement proteins, oxidative-stress metabolites, neurotrophic factors, and microRNAs; none has yet achieved sufficient analytical and clinical validation for stand-alone diagnosis. Conclusion. The most realistic precision-medicine model combines ancestry-calibrated genetic risk, established ocular phenotypes, and longitudinal molecular markers. Clinical implementation requires prospective validation, standardized assays, and evidence that biomarker-guided decisions improve visual outcomes.

KEYWORDS: aqueous humor; biomarkers; glaucoma; genetics; polygenic risk score; precision medicine.

INTRODUCTION

Primary open-angle glaucoma (POAG) is a progressive optic neuropathy characterized by retinal ganglion cell loss, remodeling of the optic nerve head, and corresponding visual-field defects. Intraocular pressure (IOP) is the only modifiable risk factor routinely targeted in clinical practice, yet glaucoma can develop at statistically normal IOP and may progress despite substantial pressure reduction. This clinical variability is consistent with a complex disease model in which aqueous-outflow resistance interacts with genetically determined neural, vascular, metabolic, and immune susceptibility [1-3].

Family aggregation and twin studies demonstrate substantial heritability, but the genetic architecture spans rare high-impact variants and a much larger number of common variants with individually small effects. Mutations in MYOC, OPTN, and TBK1 can produce Mendelian forms of open-angle glaucoma, whereas genome-wide association studies (GWAS) have mapped susceptibility across hundreds of loci [2-7]. Genetic discovery has therefore moved the field from isolated candidate genes toward pathway-level models and aggregate polygenic risk.

Parallel work in proteomics, metabolomics, cytokine profiling, and non-coding RNA analysis has generated candidate molecular biomarkers in aqueous humor, tears, and blood. These biomarkers may reflect trabecular-meshwork stress, extracellular-matrix turnover, oxidative injury, complement activation, or retinal ganglion cell degeneration [8-11]. The

central translational question is not whether measurable molecular differences exist, but whether they add reproducible clinical information beyond age, IOP, central corneal thickness, optic nerve imaging, and visual-field testing.
The aim of this review is to summarize the monogenic and polygenic architecture of POAG, critically assess molecular biomarker candidates, and define how genetic and molecular information could support risk-stratified screening, surveillance, and therapy.

MATERIALS AND METHODS

This narrative review used PubMed/MEDLINE-indexed literature available through July 2026. Search concepts included primary open-angle glaucoma, normal-tension glaucoma, MYOC, OPTN, TBK1, GWAS, multi-ancestry genetics, polygenic risk score, aqueous humor, tear biomarkers, proteomics, cytokines, microRNA, metabolomics, CRISPR, and gene therapy. We prioritized multi-ancestry association studies, systematic reviews, longitudinal clinical cohorts, and translational studies with functional validation. Evidence was organized into monogenic disease, common-variant architecture, polygenic prediction, molecular biomarker classes, and therapeutic translation. Because study selection was qualitative and no registered protocol was used, the work should be interpreted as a narrative rather than systematic review.

Monogenic forms and high-impact variants

MYOC is the most established high-impact POAG gene. Pathogenic variants account for approximately 3-4% of POAG with elevated IOP and frequently cause juvenile or early adult-onset disease [2]. Many disease-causing variants produce a toxic gain of function: misfolded myocilin accumulates in the endoplasmic reticulum of trabecular-meshwork cells, activates the unfolded-protein response, impairs aqueous outflow, and increases IOP. Penetrance varies by variant, age, and ascertainment. A meta-analysis estimated overall penetrance near 60%, but individual variants ranged from low penetrance to nearly complete expression [12]. Population data for p.Gln368Ter likewise show lower penetrance than referral-based registries, illustrating why a detected variant must be interpreted in its clinical and ancestry context [13].
OPTN and TBK1 are most strongly associated with normal-tension glaucoma. The OPTN p.Glu50Lys variant is linked to early optic nerve damage at IOP within the conventionally normal range, while TBK1 copy-number gains can produce autosomal dominant disease [3,14]. Both genes participate in autophagy, vesicular trafficking, innate immune signaling, and cellular stress responses. Their phenotypes emphasize that retinal ganglion cell vulnerability may be genetically determined independently of marked pressure elevation.
Genetic testing is most informative for early-onset disease, strong autosomal dominant pedigrees, disproportionate damage at normal IOP, or syndromic presentations. Testing should include copy-number analysis when TBK1-associated disease is suspected. A pathogenic result can prompt cascade testing and earlier surveillance of relatives, but variants of uncertain significance should not be used alone to label an asymptomatic person as affected.
The principal genes and susceptibility loci, their biological functions, and their potential clinical interpretation are summarized in Table 1 [2-7,12-16].

Table 1. Genetic components of primary open-angle glaucoma and their potential clinical interpretation

Gene or locus	Principal biological pathway	Typical association	Potential clinical relevance
MYOC	Protein folding, endoplasmic-reticulum stress, trabecular-meshwork function	Juvenile or adult POAG, often with elevated IOP	Diagnostic testing in early-onset/familial disease; cascade testing; experimental gene editing
OPTN	Autophagy, vesicular trafficking, oxidative-stress response	Familial normal-tension glaucoma, particularly p.Glu50Lys	Consider in early progressive disease with normal IOP; informs neurodegenerative mechanisms
TBK1	Autophagy and innate immune signaling; copy-number dosage	Autosomal dominant normal-tension glaucoma	Requires copy-number-aware testing; family surveillance
CDKN2B-AS1 / SIX6	Cell-cycle control, optic nerve development, retinal ganglion cell susceptibility	Common-variant POAG and optic nerve traits	Contributes to polygenic prediction; not individually diagnostic
TMCO1 / GAS7	IOP regulation and aqueous-outflow biology	Common variants associated with IOP and POAG	Supports pressure-related risk modeling
CAV1/CAV2, ABCA1, ARHGEF12	Vascular signaling, lipid transport, cytoskeleton	Population- and phenotype-dependent POAG susceptibility	Pathway discovery; limited individual predictive value
SVEP1, VCAM1, YAP1, SMAD6 and additional GWAS loci	Extracellular matrix, adhesion, mechanotransduction, vascular biology	Cross-ancestry common susceptibility	Target prioritization and polygenic models; functional validation required

As shown in Table 1, the clinical meaning of a genetic finding depends strongly on effect size and disease context. Rare pathogenic variants in MYOC, OPTN, and TBK1 may support etiological diagnosis and family testing, whereas common GWAS loci are primarily useful in aggregate polygenic models. Individual common-risk alleles should therefore not be interpreted as diagnostic findings or used alone to determine treatment [2-7,12-16].

Common-variant architecture and ancestry

Large GWAS have transformed the scale of glaucoma genetics. A 2021 multi-ethnic meta-analysis of 34,179 cases and 349,321 controls identified 127 loci, including 44 not previously reported [4]. A subsequent multitrait analysis that combined POAG, IOP, and vertical cup-to-disc ratio expanded discovery to 312 independent loci, with most signals replicating in a large external dataset [5]. These studies implicate extracellular-matrix organization, cell adhesion, lipid metabolism, vascular regulation, developmental pathways, and neuronal resilience rather than a single linear mechanism.

Ancestry is central to interpretation. A 2024 GWAS of 6,003 cases and 5,272 controls of African ancestry identified 46 significant loci and demonstrated that an African-ancestry polygenic score outperformed a score trained in a much larger European-derived dataset for African-ancestry participants [6]. This finding is clinically important because POAG burden and severe visual loss are disproportionately high in populations of African descent, while many discovery cohorts remain European enriched. Transfer of an uncalibrated score can therefore underestimate or misclassify risk.

Common loci such as CDKN2B-AS1, SIX6, TMCO1, GAS7, CAV1/CAV2, ABCA1, and ARHGEF12 influence different disease components. Some are more closely related to IOP, while others affect optic nerve morphology or ganglion-cell susceptibility [7,15,16]. A risk allele associated with disease onset does not necessarily predict the rate of visual-field progression, and lack of association in a specific cohort may reflect limited power, phenotype heterogeneity, or population-specific linkage disequilibrium [16].

Polygenic risk scores and clinical stratification

Polygenic risk scores (PRSs) aggregate effects across many variants and are the most mature route from GWAS to clinical prediction. In population and registry cohorts, individuals in the highest polygenic-risk tail had glaucoma odds comparable in magnitude to carriers of the common MYOC p.Gln368Ter risk variant [17]. In treated early POAG, high polygenic risk was associated with faster structural and visual-field progression [18]. Among individuals with established POAG, a higher PRS was associated with trabeculectomy at a younger age and a greater likelihood of bilateral surgery [19]. In ocular hypertension, PRS added information about conversion to POAG over long follow-up [20].

These findings support a future model in which genetic risk modifies the starting age and intensity of surveillance. However, PRS implementation requires ancestry-specific calibration, transparent variant weights, integration with absolute risk factors, and demonstration of clinical benefit. A high score should not automatically trigger surgery, and a low score must not override suspicious optic nerve or visual-field findings. The most defensible role is as an adjunct that refines follow-up frequency or screening priority.

Molecular biomarkers in aqueous humor, tears, and blood

Aqueous humor is anatomically proximal to the trabecular meshwork and anterior segment, but sampling is invasive and generally limited to surgery. Proteomic studies report immune activation, complement dysregulation, altered folate metabolism, and changes in selenium-related networks in POAG [8]. Cytokines and growth factors, including IL-6, IL-8, VEGF, and TGF-β-related signaling, have been studied as markers of inflammation, fibrosis, and surgical wound healing. Heterogeneity in case definition, topical medication exposure, sample volume, assay platform, and cataract-control selection limits reproducibility.

Tears are accessible and suitable for repeated sampling, but they are influenced by ocular-surface disease, preservatives, environment, and collection technique. Comparative profiling has found different cytokine patterns in tears and aqueous humor, with weak correlations between compartments [9]. This means that tears cannot be assumed to provide a noninvasive substitute for the intraocular environment without direct validation.

Oxidative-stress markers, endothelin-1, neurotrophic factors, serotonin metabolites, antioxidant capacity, and microRNAs have also been proposed [10]. MicroRNAs are attractive because they can integrate multiple pathways and may be transported in extracellular vesicles. Aqueous-humor exosomal profiling has identified glaucoma-associated microRNA signatures and linked miR-29a-3p depletion to extracellular-matrix remodeling in experimental trabecular-meshwork models [11]. These results are mechanistically informative but remain exploratory because cohorts are small and laboratory pipelines are not standardized.

The major biomarker classes, accessible specimens, biological interpretations, and current barriers to implementation are compared in Table 2 [8-11].

Table 2. Candidate molecular biomarkers in primary open-angle glaucoma

Biomarker class	Specimen	Biological interpretation	Main limitation before clinical use
Cytokines and chemokines (IL-6, IL-8, IL-12, VEGF)	Aqueous humor; tears	Inflammatory and angiogenic activity; possible wound-healing phenotype	Medication and ocular-surface confounding; inconsistent compartment correlation

Biomarker class	Specimen	Biological interpretation	Main limitation before clinical use
Complement and immune proteins	Aqueous humor	Innate immune activation and possible association with progression	Invasive sampling; heterogeneous proteomic platforms
TGF-β2 / extracellular-matrix mediators	Aqueous humor; experimental cells	Trabecular fibrosis, outflow resistance, postoperative scarring	Limited assay harmonization and threshold validation
Oxidative-stress metabolites, antioxidant capacity, endothelin-1	Tears; aqueous humor; blood	Redox imbalance, vascular dysregulation, mitochondrial stress	Systemic and preanalytical confounding; low disease specificity
BDNF and other neurotrophic markers	Tears; serum; aqueous humor	Retinal ganglion cell survival and neurodegenerative stress	Concentration depends on compartment and assay sensitivity
Cell-free and exosomal microRNAs	Aqueous humor; tears	Post-transcriptional regulation, autophagy, apoptosis, and matrix remodeling	Small discovery cohorts; normalization and isolation methods not standardized

Table 2 demonstrates that proximity to the target tissue must be balanced against feasibility of sampling. Aqueous humor may provide the most direct information about anterior-segment biology but is unsuitable for routine serial monitoring. Tears and blood are easier to obtain, although their biomarker concentrations are more vulnerable to ocular-surface, systemic, and preanalytical confounding. Consequently, no listed marker currently has sufficient validation for stand-alone clinical diagnosis [8-11].

From biomarkers to personalized treatment

Genotype may eventually define mechanistically coherent treatment groups. For MYOC-associated glaucoma, the toxic gain-of-function mechanism makes mutant-gene suppression or editing conceptually attractive. CRISPR-Cas9 disruption of MYOC reduced endoplasmic-reticulum stress and IOP in experimental systems, and lentiviral delivery has recently reproduced pressure lowering in a myocilin mouse model [21,22]. These are preclinical findings; ocular biodistribution, off-target editing, vector immunogenicity, durability, and germline safety must be addressed before human use.

A complementary strategy targets pathways downstream of genetic risk. Mitochondrial dysfunction and nicotinamide adenine dinucleotide depletion increase ganglion-cell vulnerability. Nicotinamide protected retinal ganglion cell bodies, axons, and dendrites across experimental glaucoma-relevant insults and prevented broad metabolic disruption in animal tissues [23]. Such pathway-based neuroprotection could benefit genetically diverse patients, but dosing, long-term safety, and clinically meaningful visual outcomes require randomized evaluation.

For current practice, personalized therapy remains phenotype led. Genetic and molecular data may identify patients requiring earlier surveillance or lower target IOP, but they do not yet determine whether a specific eye should receive medication, laser, or filtration surgery. The useful near-term model is layered: monogenic testing for selected families, PRS for research-supported risk refinement, and molecular panels evaluated longitudinally alongside OCT, perimetry, and IOP exposure.

A staged framework for translating these molecular findings into precision glaucoma care is presented in Table 3 [17-23].

Table 3. Proposed translational pathway for precision glaucoma care

Clinical task	Potential molecular input	Current evidence level	Required next step
Identify high-risk relatives	Pathogenic MYOC, OPTN, or TBK1 variant	Clinically actionable in selected pedigrees	Genetic counseling, segregation analysis, age-appropriate surveillance
Prioritize population screening	Ancestry-calibrated POAG PRS	Promising observational evidence	Prospective trials comparing risk-stratified and standard screening
Estimate progression risk	PRS plus OCT, visual field, IOP, age, and corneal thickness	Associations demonstrated in longitudinal cohorts	External validation and decision-curve/health-economic analysis
Monitor molecular activity	Cytokine, proteomic, metabolomic, or microRNA panel	Exploratory	Standardized collection, reference ranges, longitudinal reproducibility

Clinical task	Potential molecular input	Current evidence level	Required next step
Select mechanism-based therapy	MYOC editing; mitochondrial or neuroprotective pathway markers	Preclinical or early translational	Safety, delivery, dose, and randomized clinical efficacy studies

As summarized in Table 3, clinical readiness differs markedly across applications. Family-based testing for clearly pathogenic monogenic variants can already influence surveillance, whereas PRS-guided screening requires prospective utility studies. Molecular monitoring and mechanism-based therapy remain investigational and should be evaluated using standardized assays, external validation cohorts, and clinically meaningful visual outcomes [17-23].

Limitations and research priorities

The literature is limited by phenotype heterogeneity, underrepresentation of many ancestries, small biomarker cohorts, and inconsistent adjustment for topical therapy, surgery, age, and systemic disease. Cross-sectional differences do not establish that a biomarker predicts future damage. Multiple-testing burden and selective reporting may inflate early estimates, while technical variation in sample collection and normalization impedes replication.

Future studies should prespecify clinical use cases, enroll ancestry-diverse cohorts, and separate model development from external validation. Biomarker studies need common operating procedures for sampling, storage, assay calibration, and reporting. Genetic models should be evaluated for calibration and net clinical benefit, not only discrimination. Finally, trials must test whether biomarker-guided surveillance or treatment preserves visual function, improves quality of life, or uses resources more efficiently.

CONCLUSION

POAG comprises multiple biological routes to a common optic neuropathy. Rare variants in MYOC, OPTN, and TBK1 provide clinically meaningful diagnoses in selected families, while hundreds of common loci capture distributed susceptibility across pressure regulation, development, vascular biology, extracellular matrix, and neural survival. Polygenic scores can identify groups with earlier onset or faster progression, but ancestry portability and prospective utility remain unresolved. Molecular markers in aqueous humor, tears, and blood reveal inflammatory, metabolic, and neurodegenerative processes, yet no single analyte is ready for stand-alone diagnosis. Precision glaucoma care will most likely emerge from integrated models that combine calibrated genetic risk, longitudinal ocular phenotyping, and validated molecular panels.

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