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Research Article

Body Mass Index and Severity of Primary Open-Angle Glaucoma: A Multicenter Cross-Sectional Study in a Cameroonian Setting

Indice de Masse Corporelle et Gravité du Glaucome Primaire à Angle Ouvert : Une Etude Transversale Multicentrique Menée au Cameroun

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ABSTRACT

Background: Evidence on the association between body mass index (BMI) and primary open-angle glaucoma (POAG) severity remains limited in Sub-Saharan Africa. This study assessed this association among patients in Douala, Cameroon. **Methods:** We conducted a multicenter, hospital-based, cross-sectional analytical study from November 2024 to May 2025 in three hospitals in Douala. We included patients aged ≥35 years with confirmed POAG and reliable visual field and optical coherence tomography (OCT) results. We calculated BMI from measured height and weight. We assessed structural and functional glaucoma severity using OCT and automated perimetry. We used multivariate ordinal logistic regression to identify factors associated with severity. Statistical significance was set at $p < 0.05$. **Results:** Among 91 participants (182 eyes), mean age was 56.9 ± 13.5 years, and 56% were women. Overweight and obesity prevalences were 34.1% and 52.7%, respectively. Compared with normal-weight patients, obese patients had thinner retinal nerve fiber layer (77 ± 20 vs. 97 ± 17 µm, $p = 0.001$) and ganglion cell complex (73 ± 15 vs. 93 ± 7 µm, $p < 0.001$), larger vertical cup-to-disc ratios (0.80 ± 0.13 vs. 0.65 ± 0.16, $p = 0.001$), and worse visual field mean deviation (16 ± 7 vs. 5 ± 6 dB, $p < 0.001$). Obesity (adjusted OR [aOR] 2.74, 95% CI 1.58–4.76) and overweight (aOR 1.85, 95% CI 1.12–3.05) were independently associated with greater severity. Hypertension, older age, and higher pre-treatment intraocular pressure were also associated factors. **Conclusion:** Higher BMI was associated with greater structural and functional POAG severity. Furtherlongitudinal studies are needed to clarify this relationship.

RÉSUMÉ

Introduction : Les données sur le lien entre l'indice de masse corporelle (IMC) et la gravité du glaucome primaire à angle ouvert (GPAO) restent limitées en Afrique subsaharienne. Cette étude a évalué ce lien chez des patients de Douala, au Cameroun. **Méthodes :** Nous avons mené une étude analytique transversale multicentrique en milieu hospitalier, de novembre 2024 à mai 2025, dans trois hôpitaux de Douala. Nous avons inclus des patients âgés de 35 ans ou plus présentant un GLAO confirmé et disposant de résultats fiables du champ visuel et de la tomographie par cohérence optique (OCT). Nous avons calculé l'IMC à partir de la taille et du poids mesurés. Nous avons évalué la gravité de l'atteinte structurelle et fonctionnelle du glaucome à l'aide de l'OCT et de la périmétrie automatisée. Une régression logistique ordinale multivariée a permis d'identifier les facteurs associés à la gravité. Le seuil de signification statistique a été fixé à $p < 0,05$. **Résultats :** Parmi les 91 participants (182 yeux), l'âge moyen était de 56,9 ± 13,5 ans, et 56 % étaient des femmes. Les prévalences de surpoids et d'obésité étaient respectivement de 34,1 % et 52,7 %. Par rapport aux patients de poids normal, les patients obèses présentaient des épaisseurs de fibres nerveuses rétiniennes (77 ± 20 contre 97 ± 17 µm, $p = 0,001$) et du complexe de cellules ganglionnaires plus basses (73 ± 15 contre 93 ± 7 µm, $p < 0,001$), des rapports cup/disc verticaux plus élevés (0,80 ± 0,13 contre 0,65 ± 0,16, $p = 0,001$) et une déviation moyenne du champ visuel plus importante (16 ± 7 contre 5 ± 6 dB, $p < 0,001$). L'obésité (rapport de cotes ajusté [aOR] de 2,74, IC à 95 % : 1,58–4,76) et le surpoids (aOR de 1,85, IC à 95 % : 1,12–3,05) étaient indépendamment associés à une sévérité accrue. L'hypertension, un âge plus avancé et une pression intraoculaire plus élevée avant le traitement constituaient également des facteurs associés. **Conclusion :** Un IMC plus élevé était associé à une sévérité accrue de l'atteinte structurelle et fonctionnelle du glaucome primaire à angle ouvert. Des études longitudinales dans le futur sont nécessaires pour clarifier cette relation.

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HIGHLIGHTS FOR READERS IN A HURRY

What is already known on this topic. Primary open-angle glaucoma (POAG) is the leading cause of irreversible blindness worldwide, and Africa bears the highest burden, with an estimated prevalence of 4.2%. Elevated intraocular pressure is the only established modifiable risk factor. The contribution of body mass index (BMI) to disease severity remains inconsistent across populations, with some studies reporting a protective effect and others a harmful one. Data from sub-Saharan Africa are scarce.

T**he question this study addressed.** This hospital-based, multicentre, cross-sectional analytical study evaluated the association between BMI and POAG severity among 91 patients (182 eyes) attending three hospitals in Douala, Cameroon, between February and May 2025.

What the study adds to our knowledge. Overweight and obesity affected 34.1% and 52.7% of patients, respectively. Obese patients had significantly thinner retinal nerve fibre layer and ganglion cell complex, a larger vertical cup-to-disc ratio, and worse visual field mean deviation than normal-weight patients. After adjustment for age, sex, myopia, diabetes, hypertension, cataract, and pre-treatment intraocular pressure, obesity independently increased the odds of severe POAG 2.74-fold, and overweight 1.85-fold.

How this is relevant to clinical practice, policy or further research. These findings support incorporating weight assessment and metabolic risk counselling into routine glaucoma care and argue for longitudinal studies to clarify the causal pathways linking adiposity to glaucomatous optic nerve damage in African populations.

INTRODUCTION

Glaucoma is an optic neuropathy characterized by progressive loss of retinal ganglion cells and their axons .It is the leading cause of irreversible blindness worldwide.[1] Elevated intraocular pressure (IOP) is the most important and only modifiable risk factor identified to date, with glaucomatous damage mediated through mechanical and vascular pathways.[1] Globally, glaucoma is the second leading cause of blindness after cataract, and Africa bears the highest burden, with an incidence roughly twice the global prevalence.[2] The global prevalence of glaucoma is estimated at 2.4%, whereas in Africa, primary open-angle glaucoma (POAG) affects an estimated 4.2% of the population and primary angle-closure glaucoma 1.09%.[2] POAG is the most common subtype worldwide and accounts for 94.5% of glaucoma cases in Africa.[3],[4] In Cameroon specifically, the overall prevalence of glaucoma is estimated at 5.5%, making it the leading cause of irreversible blindness in the country, with POAG representing 4.3% of this burden.[5]

Given the devastating visual impact of POAG, identifying modifiable risk factors beyond IOP—including race, age, family history, central corneal thickness, myopia, arterial blood pressure, nutrition, education level, hematocrit level, and body mass index (BMI)—is of considerable clinical and public-health interest.[6],[7] BMI, calculated as weight in kilograms divided by height in meters squared, classifies adults as underweight (<18.5 kg/m²), normal weight (18.5–24.9 kg/m²), overweight (25.0–29.9 kg/m²), or obese (≥30.0 kg/m²) according to the World Health Organization and the U.S. Centers for Disease Control and Prevention.[8],[9] Obesity is a chronic, rapidly escalating global epidemic: nearly 2 billion people worldwide are overweight, and 650 million are obese.[11],[12] Excess adiposity has been proposed to raise IOP and glaucoma risk through inflammatory, vascular, and mechanical mechanisms, including increased episcleral venous pressure, elevated orbital fat and blood viscosity, adipocyte-derived pro-inflammatory cytokines, oxidative stress affecting the trabecular meshwork, and vascular endothelial and autonomic dysfunction impairing optic nerve perfusion.[6],[10],[13] Despite this plausible biological rationale, the epidemiological evidence linking BMI to POAG remains markedly inconsistent. A cross-sectional analysis of the Korea National Health and Nutrition Examination Survey found that not being overweight was significantly associated with POAG,[14] while a related Korean cohort found that low BMI decreased glaucoma risk only in women and in patients aged 40–49 years, and a separate Korean study found BMI associated with POAG only in men.[15],[16] A prospective analysis of the Nurses' Health Study found no significant relationship between BMI and POAG,[16] and the Barbados Eye Study found that elevated BMI was protective against POAG after adjusting for hypertension and diabetes. By contrast, a Congolese case-control study found that increased BMI was significantly associated with POAG,[17] a two-sample Mendelian randomization study reported that each standard-deviation increase in BMI raised POAG risk by approximately 90.9%,[18] and a large African-ancestry cohort study reported the opposite direction of effect, with low BMI conferring greater POAG risk and faster functional progression.[19],[20]

To date, little to no research has examined this relationship in Cameroon. This study therefore investigated the association between BMI and POAG severity, assessed through validated structural (optic disc cupping, retinal nerve fiber layer thickness) and functional (visual field mean deviation) severity measures, and IOP, among patients followed in three hospitals in Douala. Clarifying this relationship carries direct implications for early detection, risk stratification, and the potential development of targeted, cost-effective preventive strategies in a region where glaucoma-related blindness is already disproportionately high.

PATIENTS AND METHODS

Study design, setting, and duration

We conducted a multicenter, hospital-based, cross-sectional analytical study in the ophthalmology departments of three hospitals in Douala, Cameroon: the Douala General Hospital, the Second Region Military Hospital, and the Gyneco-Obstetric and Pediatric Hospital. The overall study period spanned seven months (November 2024–May 2025).

Study population and sampling

The target population comprised patients of both sexes aged 35 years and older with confirmed POAG who were

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followed up at the three study sites. We included patients with a confirmed diagnosis of POAG, at least two reliable automated visual field (AVF) results and two OCT results, and informed consent. Patients with other types of glaucoma, ocular trauma, or ocular inflammation were not included, and patients lacking the required paraclinical results or using systemic or topical steroids were excluded. The minimum sample size was calculated using Cochran's formula ($N = Z^2P(1-P)/e^2$), based on a national POAG prevalence of 4.4%,[5] a 95% confidence level ($Z = 1.96$), and a 5% margin of error, yielding a minimum requirement of 65 participants.

Data collection procedures

After administrative authorization from the three hospitals and ethical clearance, we recruited consecutive eligible patients during routine consultation hours. After obtaining informed consent, a structured questionnaire captured sociodemographic characteristics (age, sex, region of origin, occupation, residence, education), glaucoma history (duration since diagnosis, ongoing treatment, comorbidities), and presenting complaints. We measured height with a stadiometer while participants stood barefoot against a wall, and weight on a calibrated scale while participants wore light clothing and no shoes; we recorded both to calculate BMI (kg/m²).

An ophthalmologist performed a complete examination on every participant. We assessed distance visual acuity uncorrected and then corrected using the Monoyer decimal scale at 6 meters, and converted results to the logarithm of the minimum angle of resolution (LogMAR) for analysis. The anterior segment was examined by slit lamp, followed by pupillary dilation with tropicamide 0.5% and fundus examination with a 78-diopter Volk lens to assess the optic disc and cup-to-disc ratio. Intraocular pressure was measured by Goldmann applanation tonometry (normal range 10–21 mmHg), and central corneal thickness (CCT) was measured by ultrasound pachymetry integrated into the OCT device. Optical coherence tomography (Topcon Maestro®) was performed after pupillary dilation to obtain the vertical cup-to-disc ratio (VCDR), retinal nerve fiber layer (RNFL) thickness, and ganglion cell complex (GCC) thickness. Automated white-on-white static perimetry (Octopus 600®) was performed monocularly without pupillary dilation to obtain the visual field mean deviation (MD) and loss variance (LV).

Definitions and severity classification

We defined obesity as BMI ≥30 kg/m² [8]. We assessed POAG severity using structural metrics (optic nerve cupping via VCDR, RNFL, and GCC thickness on OCT) and functional metrics (visual field MD). Participants were classified into five subgroups according to Mills' classification of visual field MD: early defect (−2 to −6 dB), moderate defect (−6 to −12 dB), advanced defect (−12 to −20 dB), severe defect (<−20 dB), and end-stage defect (≤−25 dB) [21]; these were further grouped into "less severe" (early and moderate) and "more severe" (advanced, severe, end-stage) categories for analysis.

Statistical analysis

Data were analyzed using R software. We summarized qualitative variables as frequencies and percentages and quantitative variables as means ± standard deviations. We used the Kruskal-Wallis rank-sum test to compare POAG severity measures (IOP, RNFL, GCC, VCDR, visual field MD) across BMI categories (normal, overweight, obese), and Fisher's exact test where appropriate for categorical comparisons; we considered $p < 0.05$ statistically significant. We constructed a multivariate ordinal logistic regression model to estimate the independent effect of BMI category on POAG severity, adjusting for age, sex, myopia, diabetes, hypertension, cataract, and pre-treatment IOP; adjusted odds ratios (aOR) with 95% confidence intervals (CI) are reported.

Ethical considerations

The Institutional Ethics Committee of Mountain's University approved the study (Ethic Clairance No 2025/051/udm/PR/CEAQ), and the administrative directorates of the three participating hospitals authorized it. Participation was voluntary, and each participant provided informed consent after receiving complete information about the study; refusal to participate was strictly respected and did not affect care. Questionnaires were anonymized to ensure confidentiality, and collected data were used solely for the stated scientific purpose.

RESULTS

Sociodemographic and clinical characteristics

Of 200 patients screened, 91 participants (182 eyes) met the inclusion criteria .The mean age was 56.9 ± 13.5 years (range 35–90), and most participants (69.2%) were over 50 years, with the 45–54-year group most represented (27%). Women accounted for 56% of participants (n = 51) and men for 44% (n = 40), giving a female-to-male ratio of 1.28 (sex ratio M:F = 0.78). Most participants originated from the West (42.9%) and Center (26.4%) regions of Cameroon, were married (74.1%), worked in the informal sector (47.3%), had attained university-level education (63.7%), and resided in urban areas (97.8%).

A family history of glaucoma was reported by 37.4% of participants. The most frequent comorbidities were myopia (60.3%), hypertension (13.2%), and diabetes mellitus (10.1%). Glaucoma had been diagnosed less than 5 years previously in 68.1% of patients (median duration 4 years, range 1–30), and diagnosis most frequently followed a routine consultation (45.1%) rather than symptomatic presentation, with blurred vision (14.3%) and ocular pain (13.2%) being the leading presenting complaints among symptomatic patients. Bilateral visual impairment was present in 91% of patients, and the left eye was more frequently the most severely affected eye (52.7% vs. 47.3% for the right eye).

Anthropometric profile

The mean height was 1.66 ± 0.05 m and the mean weight was 83.4 ± 12.0 kg. Regarding BMI classification, overweight and obesity were highly prevalent in this POAG population: 48 participants (52.7%) were obese (moderately obese 40.7%, severely obese 11.0%, morbidly obese 1.1%), 31 (34.1%) were overweight, and only 12 (13.2%) had a normal BMI **(Table 1).**

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Table 1. BMI classification of the study population

BMI category	N=91	%
Moderately obese (30.0–34.9 kg/m²)	37	40.7
Overweight (25.0–29.9 kg/m²)	31	34.1
Severely obese (35.0–39.9 kg/m²)	10	11.0
Normal weight (18.5–24.9 kg/m²)	12	13.2
Morbidly obese (≥40.0 kg/m²)	1	1.1

Paraclinical characteristics of the overall POAG population

Pre-treatment intraocular pressure (IOP), , averaged 24.0 ± 4.3 mmHg in the right eye and 23.7 ± 4.1 mmHg in the left eye (n = 91 for each eye). Median pre-treatment IOP was 23 mmHg in both eyes; values ranged from 17 to 47 mmHg in the right eye and from 14 to 37 mmHg in the left eye. Mean IOP on treatment was 17.8 ± 4.6 mmHg in the right eye and 17.6 ± 4.5 mmHg in the left eye (Table 2). Mean visual field deviation (MD) was 11.1 ± 7.6 dB in the right eye and 12.3 ± 7.7 dB in the left eye (n = 91 for each eye). The fundoscopic cup-to-disc ratio averaged 0.63 ± 0.17 (right eye) and 0.65 ± 0.18 (left eye). On OCT, mean RNFL thickness was 84 ± 20 µm (right eye) and 82 ± 21 µm (left eye), mean GCC thickness was 77 ± 18 µm (right eye) and 75 ± 19 µm (left eye), and the vertical cup-to-disc ratio was 0.74 ± 0.16 in both eyes. Mean central corneal thickness was 515–517 µm.

Association between BMI category and POAG functional severity

Obese participants had markedly worse visual field mean deviation than normal-weight participants in both eyes (right eye: 15 ± 7 dB in obese vs. 4 ± 5 dB in normal-weight vs. 8 ± 7 dB in overweight, p < 0.001; left eye: 16 ± 7 dB in obese vs. 5 ± 6 dB in normal-weight vs. 10 ± 6 dB in overweight, p < 0.001) (Table 2). Although mean pre- and post-treatment IOP was numerically higher in obese participants than in normal-weight participants, this difference did not reach statistical significance after treatment (p = 0.9 for the right eye and p = 0.6 for the left eye).

Table 2. Comparison of intraocular pressure and functional POAG damage across BMI categories.

Parameter	Normal (N=12)	Overweight (N=31)	Obese (N=48)	p-value
IOP on treatment – RE (mmHg)	17. ± 4.6	17.5 ± 3.7	18.1 ± 5.1	0.9
IOP on treatment – LE (mmHg)	16.3 ± 3.7	17.2 ± 4.1	18.2 ± 4.9	0.6
Pre-treatment IOP – RE (mmHg)	22.5 ± 4.3	24.7 ± 5.3	24.0 ± 3.5	0.297
Pre-treatment IOP – LE (mmHg)	22.6 ± 3.7	23.7 ± 3.9	23.9 ± 4.4	0.423
Visual field MD – RE (dB)	4 ± 5	8 ± 7	15 ± 7	<0.001
Visual field MD – LE (dB)	5 ± 6	10 ± 6	16 ± 7	<0.001

IOP = intraocular pressure; RE = right eye; LE = left eye; MD = mean deviation. p-values from Kruskal-Wallis test.

Table 3. Comparison of structural POAG damage across BMI categories.

Structural parameter	Normal (N=12)	Overweight (N=31)	Obese (N=48)	p-value
OCT RNFL – RE (µm)	92±20	89±20	79±18	0.001
OCT RNFL – LE (µm)	97±17	84±22	77±20	0.001
Vertical C/D ratio – RE	0.65±0.16	0.69±0.15	0.80±0.13	0.001
Vertical C/D ratio – LE	0.68±0.14	0.68±0.18	0.80±0.14	0.002
OCT GCC – RE (µm)	93±7	78±22	73±15	<0.001
OCT GCC – LE (µm)	90±9	76±22	71±17	0.003

OCT = optical coherence tomography; RNFL = retinal nerve fiber layer; GCC = ganglion cell complex; C/D = cup-to-disc; RE = right eye; LE = left eye. p-values from Kruskal-Wallis test.

Table 4. Multivariate ordinal logistic regression: independent predictors of POAG severity.

Covariate	aOR	95% CI lower	95% CI upper	p-value
Overweight	1.85	1.12	3.05	0.017
Obese	2.74	1.58	4.76	<0.001
Age (per year)	1.06	1.02	1.10	0.003
Sex (female)	1.32	0.85	2.05	0.214
Myopia	1.45	0.92	2.29	0.109
Diabetes	1.20	0.75	1.92	0.452
Hypertension	1.67	1.10	2.54	0.016
Cataract	1.05	0.62	1.78	0.858
Pre-treatment IOP (per mmHg)	1.12	1.05	1.19	0.001

aOR = adjusted odds ratio; CI = confidence interval; IOP = intraocular pressure. Reference categories: normal BMI, male sex.

Association between BMI category and POAG structural severity

Structural damage followed the same gradient. Obese participants had significantly thinner RNFL (79 ± 18 µm right eye and 77 ± 20 µm left eye, vs. 92 ± 20 µm and 97 ± 17 µm respectively in normal-weight participants, p = 0.001 for both eyes), thinner GCC (73 ± 15 µm right eye and 71 ± 17 µm left eye, vs. 93 ± 7 µm and 90 ± 9 µm, p

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< 0.001 and p = 0.003 respectively), and a larger vertical cup-to-disc ratio (0.80 ± 0.13 right eye and 0.80 ± 0.14 left eye, vs. 0.65 ± 0.16 and 0.68 ± 0.14, p = 0.001 and p = 0.002 respectively) than normal-weight participants (Table 3). Overweight participants generally fell between normal-weight and obese participants.

Independent effect of BMI on POAG severity

In multivariate ordinal logistic regression adjusting for age, sex, myopia, diabetes, hypertension, cataract, and pre-treatment IOP, BMI category remained an independent, dose-dependent predictor of POAG severity (Table 4). Compared with normal-weight patients, overweight patients had 1.85-fold higher adjusted odds of more severe glaucoma (95% CI: 1.12–3.05, p = 0.017), and obese patients had 2.74-fold higher adjusted odds (95% CI: 1.58–4.76, p < 0.001). Older age (aOR 1.06 per year, 95% CI: 1.02–1.10, p = 0.003), hypertension (aOR 1.67, 95% CI: 1.10–2.54, p = 0.016), and each 1-mmHg increase in pre-treatment IOP (aOR 1.12, 95% CI: 1.05–1.19, p = 0.001) were independently associated with greater severity. Sex, myopia, diabetes, and cataract were not independently associated with POAG severity in the adjusted model (all p > 0.05).

Treatment modalities

Most patients (67.0%) received monotherapy, predominantly prostaglandin analogs (86.9% of monotherapy prescriptions), followed by beta-blockers (11.5%) and carbonic anhydrase inhibitors (1.6%). Dual therapy was used in 28.6% of patients, most commonly combining a prostaglandin analog with an alpha-2 agonist (34.6%) or a beta-blocker (38.5%), and triple therapy (prostaglandin analog, beta-blocker, and carbonic anhydrase inhibitor) was used in 4.4%. Treatment adherence, assessed in the subset of patients with available data, was satisfactory in 35.2% and unsatisfactory in 7.7% of the overall cohort.

DISCUSSION

The demographic profile of our cohort—mean age 56.9 years, female predominance, and diagnosis predominantly after age 50—aligns with the broader epidemiology of POAG, where risk escalates after age 40 due to cumulative IOP exposure and age-related neurodegeneration.[2],[5],[19],[22] The female predominance observed here contrasts with reports from Asian populations[16] but mirrors other African glaucoma surveys, where women more frequently access eye care services.[19],[23],[24] The over-representation of participants from the West region is consistent with described genetic susceptibility linked to myocilin mutations among Bamiléké ethnic groups in Cameroon.[5] Notably, despite high educational attainment (63.7% university level), nearly half of participants worked in the informal sector, a pattern that may reflect economic barriers to sustained glaucoma follow-up despite adequate health literacy.[20] The predominance of routine-consultation diagnosis (45.1%) over symptomatic presentation, together with advanced visual field loss already present at diagnosis in a substantial proportion of eyes, illustrates the well-described "silent thief of sight" trajectory of POAG in African populations, where substantial, often irreversible damage frequently precedes symptom onset.[19],[24]

The central finding—a graded, independent association between BMI and POAG severity—is biologically plausible and consistent with several, though not all, prior reports. Excess adiposity can raise IOP through both mechanical and vascular pathways: increased orbital fat, blood viscosity, and episcleral venous pressure elevate IOP mechanically, while impaired autonomic and endothelial function reduces choroidal and optic-nerve-head perfusion, making the nerve more vulnerable at a given IOP.[6],[13],[25] Adipose tissue additionally secretes pro-inflammatory cytokines (e.g., TNF-α, IL-6) that may promote retinal ganglion cell apoptosis, and obesity-related oxidative stress—via NADPH oxidase activation and reactive oxygen species accumulation—has been proposed to accelerate ganglion cell complex thinning, consistent with the significantly thinner GCC we observed in obese participants.[6],[18],[25] Our results are directionally consistent with a two-sample Mendelian randomization study reporting that each standard-deviation increase in BMI raised POAG risk by approximately 90.9%,[18] but contrast with a retrospective cohort from the China Health and Retirement Longitudinal Study, which found obesity to be modestly protective against glaucoma in older Chinese adults.[16] This discrepancy may partly reflect differences in outcome ascertainment (objective OCT and perimetric severity metrics in our study versus self-reported glaucoma diagnosis in the Chinese cohort) as well as population-specific differences in vascular susceptibility to obesity-related dysfunction, which has been proposed to be more pronounced in populations of African ancestry.[17],[19] It is worth noting, however, that other large cohorts of African-ancestry individuals have found the opposite association—lower BMI linked to greater POAG risk and faster progression[19],[20]—underscoring that the BMI–glaucoma relationship is unlikely to be uniform even within similarly ancestry-defined populations, and may be shaped by local patterns of comorbidity, nutrition, and disease stage at first presentation.

Limitations

Several limitations should be considered when interpreting these findings. First, the cross-sectional design precludes causal inference between BMI and POAG severity; longitudinal studies tracking BMI trajectories alongside glaucoma progression are needed to clarify temporality and causality. Second, the relatively modest sample size (91 participants, 182 eyes) limits statistical power for subgroup analyses and may not generalize to rural populations, where POAG burden and risk-factor distribution could differ substantially. Third, BMI is an indirect measure of adiposity: it does not distinguish fat mass from lean mass or indicate fat distribution, particularly central/visceral adiposity. Consequently, the observed association with BMI-defined weight categories should not be interpreted as direct evidence that visceral fat is implicated. Future studies should complement BMI with waist circumference and/or

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waist-to-hip ratio and, where feasible, body-composition measures or relevant metabolic biomarkers.

CONCLUSION

In this hospital-based cohort , elevated BMI was highly prevalent among patients with POAG and was independently associated with more advanced structural (thinner RNFL and GCC, larger vertical cup-to-disc ratio) and functional (worse visual field mean deviation) glaucomatous damage. These findings identify higher BMI as a marker associated with greater POAG severity in this population.BMI assessment may be considered as part of the broader clinical profile, alongside continued emphasis on IOP control, while further study is needed to determine the benefits of weight-management interventions for glaucoma outcomes.

DECLARATIONS

Author's contributions

All authors have contributed to the data collection and writing of this manuscript All authors read and approved the manuscript and agreed to be accountable for all aspects of the work.

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Declaration of Conflict of Interest

The authors declare that they have no conflicts of interest in relation to this study.

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