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Original article

# Study of the Relation between Primary Open Angle Glaucoma and Serum Lipid Levels

Ashraf H. El Habbak, Ahmed Saeed, Mona I. Abo Elyazeed, Mohamed Awwad

## Abstract:

**Background:** Glaucoma is identified as a progressively worsening optic neuropathy triggered by multiple factors and it is acknowledged as the foremost irreversible cause of blindness internationally. Emerging epidemiological inquiries indicate a potential association between elevated lipid levels and glaucoma, yet these findings present a dichotomy. Our research endeavor was aimed at delineating the connection between POAG and the levels of serum lipids. **Methods:** This cross-sectional, observational clinical investigation was conducted on 50 subjects in Ophthalmology Department in Benha University Hospital. Participants were classified into 25 control group and 25 POAG cases. All subjects underwent detailed history taking, best corrected visual acuity (BCVA) using Snellen's chart, Pupillary reaction, Anterior segment examination using slit lamp biomicroscopy, IOP measurement using slit lamp mounted Goldman applanation tonometer and assessment of serum lipid levels. **Results:** odd of severe POAG was 1.081 times greater for higher TC level (95%CI 1.00-1.168), 1.124 times greater for higher TG level (95%CI 1.012-1.246), and 0.569 times greater for higher VLDL level (95%CI 0.356-0.910). Univariate analysis shows that TC, TG, LDL and risk ratio I were the predictors of POAG, while multivariate analysis showed that only VLDL was the predictor of POAG. **Conclusions:** Our findings imply that dyslipidemia, characterized by high cholesterol, serum triglycerides, and LDL levels is linked to a higher risk of POAG. This research lays the groundwork for further studies on reducing dyslipidemia's impact on POAG prevalence and the effect of statin use in dyslipidemia on POAG progression.

**Keywords:** Primary Open Angle Glaucoma, Serum Lipid Levels, IOP, Severity

Ophthalmology Department, Faculty of Medicine Benha University, Egypt.

Corresponding to:
Dr. Mona I. Abo Elyazeed.
Ophthalmology Department, Faculty of Medicine Benha University, Egypt.
Email: Mona_aboelyazeed@yahoo.com

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## Introduction

Glaucoma, a condition arising from various contributing factors, leads to progressive damage to the optic nerve along with noticeable impairments in the visual field, and stands as the world's leading cause of irreversible vision loss [(1)]. The principal risk determinant in the context of glaucoma is heightened intraocular pressure (IOP). The attenuation of IOP is pivotal in safeguarding the visual field integrity among patients afflicted with glaucoma, thus constituting the paramount therapeutic paradigm in the management of this ocular ailment [(2)]. The exploration of additional risk factors is presently underway to enhance our understanding and management strategies [(3)].

Recent studies in epidemiology have indicated a potential association between hyperlipidemia and glaucoma. For instance, research conducted by A study conducted by [(4)] discovered that individuals diagnosed with hyperlipidemia exhibited a reduced risk of developing POAG compared to those not suffering from hyperlipidemia.

Nevertheless, numerous studies have also demonstrated a positive association between hyperlipidaemia and the onset of this condition. A recent comprehensive meta-analysis identified a correlation between glaucoma and elevated levels of total cholesterol, as well as decreased levels of HDL, respectively [(5)]. The augmentation of lipid concentrations in the bloodstream has been posited as a risk element for the elevation of IOP; however, the outcomes derived from studies scrutinizing this linkage have likewise exhibited a lack of consistency [(6, 7)].

The previous results further implied that the pathogenesis of various forms of glaucoma might be interconnected with alterations in hyperlipidaemia levels [(8)].

Currently, the underlying mechanisms leading to POAG remain incompletely elucidated. Hence, elucidating the relationship between hyperlipidaemia and POAG could yield valuable insights into the pathophysiological processes of this condition [(6)].

Our objective was to investigate and comprehend the linkage between POAG and serum lipid metrics, including total cholesterol, LDL, VLDL, HDL, and TG.

## Patients and Methods

This observational, cross-sectional clinical study was conducted in Ophthalmology Department in Benha University Hospital among 50 subjects during the period from 1st November 2022 to 31st October 2023. The study participants were classified into 25 control group and 25 POAG cases. Written consent was secured from all participants, who were thoroughly briefed on the study's objectives and assigned confidential identification codes.

Inclusion criteria were patients ≥18 years of age and with previously diagnosed POAG. Exclusion criteria were all types of glaucoma other than POAG, patients' refusal and patients taking lipid-lowering drugs.

**Grouping:**

Case group: included 25 primary open angle glaucoma (POAG) cases. They were sub classified into; 7 mild, 8 moderates and 10 severe cases according to mean deviation on the Humphrey 24-2 visual field analyzer as follows; mild (MD < -6 dB), moderate (MD -6 to -12 dB) and severe (MD > -12 dB), and control group: included 25 normal subjects.

POAG was identified through the presence of increased intraocular pressure (IOP > 21 mmHg), evidence of glaucomatous damage to the optic nerve, and accompanying visual field (VF) deterioration. VF impairment was evaluated by detecting a group of three or more adjoining, non-peripheral points on the pattern deviation plot that remained clear of the horizontal meridian. These points demonstrated a probability of less than 5% of appearing in a comparable age group in the general population, with a

minimum of one point showing a probability of less than 1%. Additionally, the observed pattern had to demonstrate an abnormal standard deviation, with a probability of less than 0.05 of occurring in the general population, and meet specific test reliability standards: fixation losses below 20%, false positives under 15%, and false negatives below 15% [9, 10].

Each subject within control cohort was subjected to initial ophthalmological assessments, encompassing evaluations of refractive condition, gonioscopy, and examination via slit lamp biomicroscopy, all conducted by specialists in glaucoma. The criteria for including normal participants were specified as follows: IOP $\leq$ 21 mmHg, no evidence of glaucomatous optic neuropathy based on clinical examination, no glaucomatous visual field defects, and no family history of POAG in first degree relatives.

All studied cases were subjected to detailed history taking, including personal history (age, sex, occupation, special habits), past medical history (diabetes mellitus, hypertension, COPD), past surgical history (history of any surgical procedures), and previous medications.

Best corrected visual acuity (BCVA) using Snellen's chart, Pupillary reaction, Anterior segment examination using slit lamp biomicroscopy. Intraocular pressure measurement using slit lamp mounted. Goldman applanation tonometer (Haag-Streit International, Köniz, Switzerland). Dilated fundus examination using indirect ophthalmoscope and the 90 diopter Volk lens. Gonioscopy by 3 Mirror Zeiss Gonio lens was done to assess Angle grade using the Shaffer's grading system and exclude other causes of glaucoma. Visual field perimetry SITA Standard 24-2 using the Humphrey Field Analyzer 750 (Humphrey-Zeiss Instruments, Dublin, CA).

**Assessment of serum lipid and lipoprotein concentrations:**

The quantification of serum lipid levels, including TC, HDL, LDL, and TG, was executed utilizing biochemical analytical techniques on samples obtained at the initial collection point (ILab, Milano, Italy). The criteria set forth by the National Cholesterol Education Program (NCEP) Adult Treatment Panel III (ATP III) were adhered to for the interpretation of lipid concentrations, wherein hypercholesterolemia is characterized by a total cholesterol level exceeding 200 mg/dl, hypertriglyceridemia is identified with triglyceride levels surpassing 150 mg/dl, LDL concentrations above 130 mg/dl are deemed elevated, and HDL measurements below 40 mg/dl are considered deficient [11].

**Sample size:**

The calculation of the sample size was based on the formula: $n = Z^2 * P * (1 - P) / d^2$, where n represents the sample size, Z denotes the Z statistic corresponding to a specified confidence level, P is the expected prevalence according to prior studies, and d signifies the level of precision. Assuming a Z value of 1.96 for a 95% confidence interval, P as 0.024 (2.4%), and d as 5% (0.05), the calculated minimum sample size required is 36.

**Statistical analysis:**

The gathered data were documented, followed by presentation and statistical analysis conducted on a computer with SPSS version 27.0 for Windows (SPSS Inc., Chicago, IL, USA) as follows: The normality of distribution for the analysed variables was tested using Kolmogorov-Smirnov test. All numerical data were non-normally distributed. The collected data were summarized in terms of median and inter-quartile range (IQR) for quantitative data and as number and percentage for qualitative data. For categorical variables, comparisons between the different study groups were carried out using Fisher's Exact Test (FET). Kruskal Wallis Test was used to compare differences between more than two groups for non-normally distributed quantitative variables. Correlation analysis to determine the association between variables was done

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using Kendall's tau-b. Multinomial logistic regression was used to detect the odds ratio of different groups with other demographic, ocular and lipid profile variables.

Binary logistic regression analysis was employed to identify factors predicting the development of POAG. All statistical tests conducted were bidirectional. In this study, a significance threshold was established at ($p \leq 0.05$).

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## Results

This table illustrates that no statistically significant difference was observed between mild, moderate, severe and control groups as regard their age. Moreover, partial eta squared showed moderate effect size. In addition, no significant difference was exhibited between the four groups as regarding gender nor previous medical history ($p>0.05$). This table illustrates that a statistically significant difference was observed between groups ($p=0.023$) as regard BCVA, Patients with severe POAG showed the lowest median BCVA (0.3) across different groups. Moreover, partial eta squared showed large effect size the table demonstrates that there was a statistically significant difference between groups regarding IOP. Moreover, partial eta squared showed large effect size, C/D ratio significantly was the highest among patients with severe POAG (0.9), and lowest among control group (0.4). Moreover, partial eta squared showed large effect size. POAG severe patients significantly showed the highest VF (-25.1) in comparison with the other 3 groups. Moreover, partial eta squared showed large effect size **Error! Reference source not found.**. This table shows that there was a statistically significant higher median TC level among patients with severe POAG (246.5) in comparison with other 3 groups ($p<0.001$). Similarly, patients with severe POAG had highest TG level among all groups ($p<0.001$). No statistically significant difference was observed between the different groups as regard HDL, LDL, and VLDL Table 2.

**Table 1:** Demographic and previous medical history, BCVA, C/D ratio among the studied groups

| Variable (n=50) | | Mild cases (n=7) | | Moderate cases(n=8) | | Severe cases(n=10) | | Control group(n=25) | | p-value |
|---|---|---|---|---|---|---|---|---|---|---|
| | | No | % | No | % | No | % | No | % | |
| Age Median (IQR) | | 60.0 (52.066.0) | | 54.0 (43.8- 61.3) | | 52.0 (50.0-63.5) | | 51.0 (45.5-60.5) | | 0.372* |
| Gender | Male | 5 | 71.4 | 4 | 50.0 | 3 | 30.0 | 11 | 44.0 | 0.430** |
| | Female | 2 | 28.6 | 4 | 50.0 | 7 | 70.0 | 14 | 56.0 | |
| History | DM | 3 | 42.9 | 2 | 25.0 | 1 | 10.0 | 3 | 12.0 | |
| | HTN | 0 | 0.0 | 1 | 12.5 | 0 | 0.0 | 2 | 80.0 | |
| | DM & HTN | 2 | 28.6 | 0 | 0.0 | 0 | 0.0 | 2 | 8.0 | 0.096** |
| | COPD | 0 | 0.0 | 0 | 0.0 | 1 | 10.0 | 0 | 0.0 | |
| | NAD | 2 | 28.6 | 5 | 62.5 | 8 | 80.0 | 18 | 72.0 | |
| | | Median | IQR | Median | IQR | Median | IQR | Median | IQR | |
| BCVA | | 0.6 | 0.6-0.7 | 0.6 | 0.6-0.9 | 0.3 | 0.1-0.5 | 0.6 | 0.4-0.7 | **0.023(S)** |
| IOP (mmHg) on treatment | | 18.0 | 13.0-20.0 | 19.5 | 12.0-20.8 | 23.0 | 21.0-30.5 | 17.0 | 15.0-18.0 | **<0.001** |
| C/D Ratio | | 0.6 | 0.5-0.7 | 0.6 | 0.5-0.8 | 0.9 | 0.8-0.9 | 0.4 | 0.3-0.5 | **<0.001** |
| VF (MD) | | -4.3 | 5.5/-3.5 | -8.7 | 10.1/-7.0 | -25.1 | 27.7/19.7 | -0.9 | 1.3/-0.7 | **<0.001** |

IQR=Interquartile Range (Percentile25-Percentile75), DM : Diabetes mellites, HTN : Hypertension, COPD: Chronic obstructive pulmonary disease, IOP= Intraocular pressure measure in mmHG, VF=Visual field NAD: No abnormalities detected. *Kruskal-Wallis test **Fisher Exact test***Partial eta squared ****Camer V effect size

**Table 2:** Lipid profile among the studied groups.

| Variable (n=50) | Mild cases (n=7) Median | IQR | Moderate cases (n=8) Median | IQR | Severe cases (n=10) Median | IQR | Control group (n=25) Median | IQR | p-value* |
|---|---|---|---|---|---|---|---|---|---|
| **TC** | 184.0 | 163.0-246.0 | 197.5 | 179.3-249.8 | 246.5 | 217.3-351.0 | 138.0 | 119.5-172.0 | **<0.001 (HS)** |
| **TG** | 90.0 | 35.0-121.0 | 62.0 | 44.8-118.8 | 176.5 | 135.3-239.8 | 59.0 | 40.5-98.0 | **<0.001 (HS)** |
| **HDL** | 40.0 | 39.0-51.0 | 60.5 | 43.0-64.5 | 68.0 | 37.5-102.5 | 51.0 | 41.0-67.5 | 0.284 |
| **LDL** | 127.4 | 10.2-4-162.0 | 126.4 | 104.0-192.9 | 136.0 | 112.3-150.4 | 92.1 | 77.5-122.8 | 0.053 |
| **VLDL** | 16.6 | 7.0-23.6 | 12.1 | 9.0-23.0 | 17.4 | 11.6-26.5 | 14.6 | 9.6-23.3 | 0.865 |
| **Risk ratio I** | 3.8 | 3.2-6.3 | 4.2 | 2.9-4.5 | 3.5 | 2.6-6.3 | 2.7 | 2.4-3.2 | **0.004 (HS)** |
| **Risk Ratio II** | 2.6 | 2.0-4.3 | 3.0 | 1.8-3.2 | 1.9 | 1.2-3.8 | 1.7 | 1.5-2.2 | 0.096 |

TC: Total cholesterol, TG: Triglycerides, HDL: High-density lipoprotein, LDL: Low-density lipoprotein, VLDL: Very low-density lipoprotein. * Kruskal Wallis test

There was a statistically significant weak positive association between severity of cases and TC level (r=0.363, p0.024), and moderate positive association with TG level (r=0.479, p=0.003) Table 3.

Table 4 shows the multinomial analysis by demographic and previous medical history. Patients who suffered from both DM and HTN had 62.620 higher likelihood of being with mild symptoms (95%CI 1.095-647.445). odd of severe POAG was 1.081 times greater for higher TC level (95%CI 1.00-1.168), 1.124 times greater for higher TG level (95%CI 1.012-1.246), and 0.569 times greater for higher VLDL level (95%CI 0.356-0.910) Severe cases showed higher odds in comparison with the other groups in BCVA, and IOP (P<0.05). Also, mild cases had 14.368-fold of C/D ratio with 95%CI 12.36116.700, while moderate cases were 19.113-fold and severe cases was 99.622-fold and the relation was significant (P<0.05).

This table illustrates that odd of severe POAG was 1.081 times greater for higher TC level (95%CI 1.00-1.168), 1.124 times greater for higher TG level (95%CI 1.012-1.246), and 0.569 times greater for higher VLDL level (95%CI 0.356-0.910). Univariate analysis shows that TC, TG, LDL and risk ratio I were the predictors of POAG, while multivariate analysis showed that only VLDL was the predictor of POAG Table 5.

**Table 3:** Correlation between Severity of cases and lipid profile among studied cases (n=25)

| Variables (n=25) | | Severity of cases |
|---|---|---|
| **TC** | **Correlation coefficient** | 0.363 |
| | **p-value** | **0.024 (S)** |
| **TG** | **Correlation coefficient** | 0.479 |
| | **p-value** | **0.003 (HS)** |
| **HDL** | **Correlation coefficient** | 0.280 |
| | **p-value** | 0.084 |
| **LDL** | **Correlation coefficient** | 0.036 |
| | **p-value** | 0.822 |
| **VLDL** | **Correlation coefficient** | 0.101 |
| | **p-value** | 0.532 |
| **Risk ratio I** | **Correlation coefficient** | -0.145 |
| | **p-value** | 0.368 |
| **Risk Ratio II** | **Correlation coefficient** | -0.193 |
| | **p-value** | 0.230 |

TC: Total cholesterol, TG: Triglycerides, HDL: High-density lipoprotein, LDL: Low-density lipoprotein, VLDL: Very low-density lipoprotein. Kendall's tau-b correlation

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**Table 4:** Relationship between patients' demographics and likelihood of having more severe primary open angle glaucoma in multinomial logistic regression model.

| Variables (n=50) | | Mild Vs. Control OR (95%CI) | p-value | Moderate Vs. Control OR (95%CI) | p-value | Severe Vs. Control OR (95%CI) | p-value |
|---|---|---|---|---|---|---|---|
| **Age** | | 1.005 (0.8701.161) | 0.946 | 0.968 (0.8711.075) | 0.542 | 1.063 (0.9491.190) | 0.293 |
| **Gender** | **Female** | 1.00 (Reference) | | 1.00 (Reference) | | 1.00 (Reference) | |
| | **Male** | 9.159 (0.459182.689) | 0.147 | 1.674 (0.194-14.444) | 0.542 | 0.329 (0.0392.803) | 0.309 |
| **History** | **NAD** | 1.00 (Reference) | | 1.00 (Reference) | | 1.00 (Reference) | |
| | **COPD** | 11.970 (11.97-11.970) | 1.000 | 1.519 (1.5191.519) | 1.000 | 11.379 (0.000-25.364) | 0.998 |
| | **DM** | 12.220 0.802186.198) | 0.072 | 3.303 (0.331-32.990) | 0.309 | 0.414 (0.0295.919) | 0.516 |
| | **HTN** | 0.000 (0.0000.000) | 0.996 | 1.441 (0.087-23.784) | 0.798 | 0.000 (0.0000.000) | 0.996 |
| | **DM&HTN** | 62.620 (1.095647.445) | **0.044 (S)** | 0.000 (0.0000.000) | 0.997 | 0.000 (0.0000.000) | 0.996 |
| **TC** | | 1.054 (0.977-1.137) | 0.171 | 1.044 (0.9941.096) | 0.088 | 1.081 (1.0001.168) | **0.049 (S)** |
| **TG** | | 1.041 (0.949-1.142) | 0.391 | 1.042 (0.9691.119) | 0.267 | 1.124 (1.0141.246) | **0.026 (S)** |
| **HDL** | | 0.879 (0.765-1.010) | 0.069 | 0.995 (0.9061.093) | 0.920 | 0.942 (0.8331.066) | 0.344 |
| **LDL** | | 0.991 (0.913-1.072) | 0.817 | 1.001 (0.9511.055) | 0.959 | 0.940 (0.8541.034) | 0.206 |
| **VLDL** | | 0.721 (0.464-1.120) | 0.146 | 0.793 (0.5501.144) | 0.215 | 0.569 (0.3560.910) | **0.019 (S)** |
| **BCVA** | | 6.592 (0.114380.220) | 0.362 | 14.050 (0.259763.595) | 0.195 | 0.019 (0.0010.642) | **0.027 (S)** |
| **IOP (mmHg) on treatment** | | 1.050 (0.8001.377) | 0.725 | 1.103 (0.8521.428) | 0.456 | 1.750 (1.2012.549) | **0.004 (HS)** |
| **C/D Ratio** | | 14.368 (12.36116.700) | **0.006 (HS)** | 19.113 (17.12021.340) | **0.005 (HS)** | 99.622 (29.057341.150) | **<0.001 (HS)** |
| **VF (MD)** | | 4.507 (0.0006.367) | 0.967 | 7.116 | 0.958 (0.0001.989) | 1.878 (0.0002.639) | 0.957 |

OR=Odds ratio, CI=Confidence interval, HTN: Hypertension, COPD: Chronic obstructive pulmonary disease, IOP: Intraocular pressure measure in mmHg, VF=Visual field, TC: Total cholesterol, TG: Triglycerides, HDL: High-density lipoprotein, LDL: Low-density lipoprotein, VLDL: Very low-density lipoprotein

**Table 5:** Univariate and multivariate logistic regression analysis

| Variable (n=50) | Univariate logistic regression | | | Multivariate logistic regression | | |
|---|---|---|---|---|---|---|
| | Crude OR | 95%CI | p-value | Adjusted OR | 95%CI | p-value |
| **TC** | 0.961 | 0.939-0.984 | <0.001 (HS) | 0.857 | 0.698-1.054 | 0.144 |
| **TG** | 0.986 | 0.976-0.997 | 0.012 (S) | 0.933 | 0.869-1.003 | 0.059 |
| **HDL** | 0.987 | 0.960-1.015 | 0.372 | 1.292 | 0.927-1.800 | 0.131 |
| **LDL** | 0.980 | 0.964-0.996 | 0.017 (S) | 1.064 | 0.850-1.331 | 0.589 |
| **VLDL** | 1.004 | 0.954-1.056 | 0.879 | 1.447 | 1.012-2.069 | 0.043 (S) |
| **Risk ratio I** | 0.406 | 0.200-0.822 | 0.012 (S) | 125.26 6 | 0.060-260.719 | 0.215 |
| **Risk Ratio II** | 0.650 | 0.373-1.132 | 0.128 | 0.072 | 0.000-914.104 | 0.585 |

TC: Total cholesterol, TG: Triglycerides, HDL: High-density lipoprotein, LDL: Low-density lipoprotein, VLDL: Very low-density lipoprotein.

## Discussion

Concerning the risk factors identified in the current research, it was found that there was a statistically significant higher median TC and TG level among patients with severe POAG in comparison with other 3 groups (p<0.001). There was statistically significant weak positive association between severity of cases and

TC level (r=0.363, p0.024), and moderate positive association with TG level (r=0.479, p=0.003).

Numerous investigations have inadequately considered critical confounders in the relationship between lipid concentrations and intraocular pressure IOP, such as BMI [12], diabetic condition [13],or the usage of pertinent medications like statins [12-14]. These factors potentially bear correlations with IOP [15].

Moreover, there has been a notable absence of research specifically investigating the relationship with IOPcc. By exploring the correlation between serum lipid concentrations and IOPcc, our investigation aimed to minimize confounding factors linked to lipid interactions with corneal biomechanical characteristics. For instance, there is indication that individuals with diabetes—a condition frequently correlated with hyperlipidemia—exhibit increased corneal mechanical stiffness, which may be related to artificially inflated IOP readings [16].

The physiological interconnectivity between cholesterol fractions and serum lipid concentrations complicates the discernment of significant correlations with any singular component. Remarkably, the correlations with IOPcc either remained largely consistent or were only slightly diminished (yet continued to be significant). Given the recognized variation in lipid levels across genders, analyses stratified by sex were conducted, revealing no alterations in the direction or statistical significance of the associations when adjusted for multiple variables. An essential interaction between gender and each lipid fraction was observed, with men displaying a more pronounced correlation, especially in relation to HDL-C [17].

Additionally, a specific SNP located near the ABCA1 gene (rs2487032) has been linked to the high-tension POAG endophenotype, characterized by primary open-angle glaucoma with an IOP exceeding 22 mmHg[18].

Links to IOP have also been established with polymorphisms at the CAV1/2 gene loci (rs10281637), which are responsible for encoding caveolin 1 and caveolin 2 proteins. These proteins are believed to be involved in lipid metabolism [19]. Caveolin 1, a crucial element of caveolae, participates in a variety of cellular processes such as transport, signal transduction, and the management of cholesterol metabolism. Intriguingly, it has been discovered that ABCA1 influences caveolin 1 concentrations through mechanisms that might also contribute to the regulation of IOP [20].

Despite the absence of a shared biochemical progenitor for HDL-C and LDL-C and their functionally divergent roles within the pathway of atherogenesis, the homogeneity in their structural surface proteins, specifically apolipoproteins, might elucidate their concurrent positive correlations with IOP. For instance, apolipoprotein B, which predominantly marks LDL-C particles, is pivotal in the transport of lipids. Earlier investigations have demonstrated that elevated levels of apolipoprotein B correlate with increased IOP. In a similar vein, apolipoprotein A, the principal protein constituent of HDL-C, has likewise been previously linked with augmented IOP, particularly in male subjects [13].

Previous results elucidated that their investigations delineate a significant correlation between elevated serum lipid concentrations and an augmented risk of POAG. The average levels of cholesterol, triglycerides, and LDL were significantly elevated in the case group versus the control group, ascertained with a 95% CI, whereas the variance in HDL levels between the two groups did not present a statistically significant discrepancy [21].

The previous results underscored the profound linkage between elevated levels of cholesterol, LDL, and triglycerides, alongside reduced HDL, and their association with POAG. Although HDL levels were observed to be lower in

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individuals with POAG compared to the control group, this variance did not achieve statistical significance. Additionally, LDL concentrations were significantly higher in cases compared to controls [22].

Findings indicated that High cholesterol levels were present in 27.5% (n=11), high triglyceride in 42.5% (n=17), low HDL levels were present in 40% (n=16), High LDL levels were observed among 30% (n=12) of participants and high VLDL in 57.5% (n=23). The differences in the distribution of different grades of POAG with respect to different levels of LDL and total cholesterol were not significant, with p-values of 0.202 and 0.123 respectively. However, it was statistically significant for low HDL, high triglycerides and high VLDL with p value of 0.004, 0.05 and 0.017 respectively [23].

In the present study, it was found that patients who suffered from both DM and HTN had 62.620 higher likelihood of being with mild symptoms (95%CI 1.095-647.445).

Findings of previous studies posited that the interplay of diabetes and hypertension, whether independently or in conjunction, plays a significant role in the manifestation of glaucoma. Interestingly, isolated dyslipidemia was observed to marginally reduce the risk of glaucoma development by approximately 5%. Nonetheless, when dyslipidemia coexists with either diabetes or hypertension, there is a notable escalation in the risk profile [4, 22].

The findings suggest a synergistic influence of dyslipidemia alongside hypertension or diabetes in the pathogenesis of glaucoma. Our investigation reveals dyslipidemia as an autonomous risk factor for glaucoma, after the exclusion of these confounding variables.

In the present study regarding univariant analysis it was found TC, TG, LDL, and risk ratio I were the predictors of POAG, while multivariate analysis showed that only VLDL was the predictor of POAG. It was highlighted that Univariate analyses found that higher levels of TC and LDL-C were associated with higher IOPcc in both cohorts and that higher triglyceride levels were associated with higher IOPcc only in the UK Biobank cohort [17]. In the initial univariate analyses across both study groups, high-density lipoprotein cholesterol (HDL-C) demonstrated no link with intraocular pressure corrected for central corneal thickness (IOPcc). However, this relationship transformed into a significant positive association within both cohorts upon the integration of gender and age variables into multivariate analyses. Noteworthy were the substantial interactions detected between gender and each lipid marker (with P-values of 0.003, <0.001, 0.049, and 0.003, respectively, for TC, HDL-C, LDL-C, and triglycerides), indicating a more pronounced linkage in males, particularly in the context of HDL-C. In the realm of multivariate scrutiny, after accounting for a spectrum of confounders such as demographic attributes, lifestyle preferences, cardiovascular risk determinants, and prevalent usage of cardiovascular drugs, elevated quantities of TC, HDL-C, and LDL-C were found to correlate with higher IOPcc across both populations studied. Contrastingly, an increase in triglyceride levels was found to inversely relate to IOPcc.

## Conclusions:

Dyslipidemia, recognized as a contributory factor for myriad health conditions, is implicated by our research as being linked with a heightened risk of POAG, as evidenced by associations with elevated total cholesterol, serum triglycerides, and LDL levels. This investigation paves the way for future research endeavors, including exploring the impact of dyslipidemia mitigation on reducing IOP and the incidence of POAG, as well as examining the influence of statin usage for dyslipidemia management on the progression of POAG.

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