RESEARCH PAPER

Correlation between Serum Lipid Levels and Primary Open-Angle Glaucoma-A Case Control study

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ABSTRACT

Background: Primary open-angle glaucoma (POAG) is a leading cause of irreversible blindness worldwide. Increasing evidence suggests that systemic metabolic factors such as dyslipidemia may contribute to its pathogenesis.

Aim: To evaluate the association between serum lipid levels and primary open-angle glaucoma.

Methods: This hospital-based observational case–control study included 84 participants (42 POAG cases and 42 age- and gender-matched controls). All participants underwent comprehensive ophthalmic examination and fasting serum lipid profiling. Statistical analysis was performed using SPSS version 22. Independent t-test and Chi-square test were applied.

Results: Among the 42 POAG cases, 25 (59.5%) were males and 17 (40.5%) were females, while among the 42 controls, 26 (61.9%) were males and 16 (38.1%) were females. There was no significant difference in gender distribution between the two groups (p = 1.00). Serum triglycerides, total cholesterol, LDL, and VLDL levels were significantly higher in POAG patients, while HDL levels were significantly lower compared to controls

Conclusion: POAG is associated with an atherogenic lipid profile. Dyslipidemia may play a role in glaucomatous optic neuropathy.

Keywords: Primary open-angle glaucoma, POAG, Serum lipids, Dyslipidemia, Cholesterol, Triglycerides, Case-control study.

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BACKGROUND

Primary open-angle glaucoma (POAG) is a chronic progressive optic neuropathy characterized by retinal ganglion cell loss, optic nerve head cupping, and corresponding visual field defects in the presence of an open anterior chamber angle. It is one of the leading causes of irreversible blindness worldwide.[1-3]

Although elevated intraocular pressure (IOP) remains the most important modifiable risk factor, it alone cannot fully explain the development of the disease.[4] Many patients continue to progress despite adequate IOP control, suggesting the role of pressure-independent mechanisms such as vascular dysregulation, oxidative stress and impaired optic nerve head perfusion.[5,6]

Dyslipidemia, characterized by elevated total cholesterol, triglycerides, LDL, and VLDL, along with reduced HDL, contributes to endothelial dysfunction and microvascular compromise.[4,7] Reduced nitric oxide bioavailability and oxidative stress may impair optic nerve perfusion and contribute to glaucomatous damage.[6,8]

Although several studies have evaluated the association between serum lipid levels and primary open-angle glaucoma, the findings remain inconsistent and inconclusive.[9-13] Furthermore, limited data are available from the Indian population, particularly in the South Indian context. Hence, the present study was undertaken to evaluate the correlation between serum lipid levels and primary open-angle glaucoma.

MATERIALS AND METHODS

This observational case–control study was conducted at R.L. Jalappa Hospital and Research Centre, Kolar, from April 2024 to November 2025. A total of 84 participants were included in the study, comprising 42 patients diagnosed with primary open-angle glaucoma (POAG) and 42 age- and gender-matched controls.

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Patients aged between 40 and 80 years diagnosed with POAG, characterized by open anterior chamber angles on gonioscopy, glaucomatous optic disc changes, and corresponding visual field defects, were included in the study.

The control group included individuals with intraocular pressure less than 21 mmHg, normal optic discs, and no evidence of glaucomatous visual field defects.

Patients with diabetes mellitus, hypertension, thyroid disorders, cardiovascular disease, high myopia (> −3D), pseudoexfoliation, and those on lipid-lowering therapy were excluded from the study.

All participants underwent a comprehensive ophthalmic evaluation, including best corrected visual acuity assessment, slit-lamp examination, Goldmann applanation tonometry, gonioscopy, optic disc evaluation, and Humphrey visual field analysis.

After an overnight fasting, venous blood samples were collected from all participants. Serum lipid levels , including total cholesterol, triglycerides, high-density lipoprotein (HDL), low-density lipoprotein (LDL), and very low-density lipoprotein (VLDL), were measured using an automated biochemical analyzer.

RESULTS

Table 1. Age group distribution of study participants in POAG cases and controls

Age group (years)	POAG cases	Controls
41–50	3	1
51–60	12	13
61–70	17	20
71–80	10	8
Total	**42**	**42**
Mean ± SD	**63.93 ± 9.52**	**64.90 ± 8.07**
p-value = 0.52		

The age group distribution was comparable between POAG cases and controls, with the majority of participants in both groups belonging to the 61–70 year age category. No statistically significant difference was observed in age group distribution between the two groups, indicating adequate age matching for subsequent comparative analyses.

Table 2. Gender distribution of study participants in POAG cases and controls

Sex	POAG cases	Controls
Male	25	26
Female	17	16
Total	**42**	**42**
p-value = 1.00		

The gender distribution was comparable between POAG cases and controls, with a male predominance observed in both groups. No statistically significant difference was noted, indicating adequate sex matching between the study groups.

Table 3. Comparison of total cholesterol classification and mean levels between POAG cases and controls

Total cholesterol classification	POAG cases	Controls	Total
Desirable (<200 mg/dL)	21	35	56
Borderline high (200–239 mg/dL)	16	7	23
High (≥240 mg/dL)	5	0	5
Mean ± SD (mg/dL)	**211.4 ± 34.6**	**192.8 ± 29.9**	—
p-value = 0.003			

Total cholesterol distribution differed significantly between POAG cases and controls. POAG cases showed a higher frequency of borderline high and high cholesterol categories, along with a significantly higher mean total cholesterol level compared to controls, indicating an increased dyslipidaemic burden among POAG patients.

Table 4. Comparison of triglyceride classification and mean levels between POAG cases and controls

Triglycerides classification	POAG cases	Controls	Total
Normal (<150 mg/dL)	10	22	32
Borderline high (150-199mg/dL)	13	15	28
High (>200 mg/dL)	19	5	24

Mean ± SD (mg/dL)	**185.10 ± 47.91**	**153.12 ± 44.49**	-
p-value < 0.001			

Triglyceride distribution showed a statistically significant difference between POAG cases and controls. Higher triglyceride categories were more frequently observed among POAG cases, along with a significantly higher mean triglyceride level, indicating a greater prevalence of hypertriglyceridaemia in the POAG group.

Table 5. Comparison of HDL cholesterol classification and mean levels between POAG cases and controls

HDL cholesterol classification	POAG cases	Controls	Total
Low (<40 mg/dL)	25	8	33
Normal (40–59 mg/dL)	16	31	47
High / Protective (≥60 mg/dL)	1	3	4
Mean ± SD (mg/dL)	**37.24 ± 11.09**	**46.38 ± 10.20**	—
p-value <0.001			

A statistically significant difference in HDL cholesterol distribution was observed between POAG cases and controls. Low HDL cholesterol levels were more prevalent among POAG cases, accompanied by a significantly lower mean HDL level compared to controls, suggesting an unfavorable lipid profile in the POAG group.

Table 6. Comparison of LDL cholesterol classification and mean levels between POAG cases and controls

LDL cholesterol classification	POAG cases	Controls	Total
Optimal (<100 mg/dL)	8	11	19
Near optimal (100–129 mg/dL)	8	9	17
Borderline high (130–159 mg/dL)	11	20	31
High (160–189 mg/dL)	10	0	10
Very high (≥190 mg/dL)	5	2	7
Mean ± SD (mg/dL)	**146.8 ± 36.9**	**121.6 ± 29.4**	—
p-value = 0.002			

LDL cholesterol distribution differed significantly between POAG cases and controls. Higher LDL categories, including high and very high levels, were more frequent among POAG cases, along with a significantly higher mean LDL cholesterol level, indicating greater atherogenic lipid burden in the POAG group.

Table 7. Comparison of VLDL cholesterol classification and mean levels between POAG cases and controls

VLDL cholesterol classification	POAG cases	Controls	Total
Normal (5–40 mg/dL)	25	37	62
High (>40 mg/dL)	17	5	22
Mean ± SD (mg/dL)	**37.36 ± 9.57**	**31.02 ± 8.76**	—
p-value = 0.002			

A statistically significant difference in VLDL cholesterol distribution was observed between POAG cases and controls. Elevated VLDL levels were more common among POAG cases, with a significantly higher mean VLDL concentration compared to controls, indicating an adverse lipid profile in the POAG group.

Table 8. Comparison of biochemical parameters between POAG cases and controls

Parameter	POAG cases	Controls	p-value
Triglycerides (mg/dL)	185.10 ± 47.91	153.12 ± 44.49	0.002
Total Cholesterol (mg/dL)	197.31 ± 43.45	165.17 ± 32.60	< 0.001
HDL (mg/dL)	37.24 ± 11.09	46.38 ± 10.20	< 0.001
LDL (mg/dL)	143.74 ± 40.43	122.81 ± 31.35	0.010
VLDL (mg/dL)	37.36 ± 9.57	31.02 ± 8.76	0.002

A statistically significant difference was observed between POAG cases and controls for all assessed biochemical parameters. POAG cases had significantly higher mean levels of triglycerides, total cholesterol, LDL, and VLDL, along with significantly lower HDL levels compared to controls, indicating a more atherogenic lipid profile among POAG patients. Overall, these findings demonstrate clear biochemical differences between POAG cases and controls, supporting the presence of an unfavorable lipid profile in the POAG group.

POAG patients demonstrated significantly higher atherogenic lipid fractions and significantly lower HDL levels compared to controls.

DISCUSSION

This case–control study included 42 patients with primary open-angle glaucoma (POAG) and 42 age- and gender-matched controls, with no statistically significant difference in age ($p = 0.52$) or gender distribution ($p = 1.00$). This comparability between groups reduces confounding and strengthens the validity of the observed biochemical differences.

The present study demonstrated that patients with POAG exhibited an atherogenic lipid profile, characterized by significantly higher levels of total cholesterol, triglycerides, low-density lipoprotein (LDL), and very low-density lipoprotein (VLDL), along with significantly lower levels of high-density lipoprotein (HDL).

Elevated total cholesterol levels observed in POAG patients in this study are consistent with previous findings. In a case–control study, Pavljasević and Asćerić reported significantly higher serum cholesterol levels in patients with POAG compared to controls.[11] Similarly, Karn et al., in a recent clinical observational study, demonstrated increased levels of total cholesterol and other atherogenic lipid fractions in glaucoma patients.[13] A meta-analysis by Huang et al. reported a positive association between elevated cholesterol levels and the risk of POAG, although with considerable heterogeneity across studies.[9]

Triglyceride levels were also significantly elevated in POAG patients in the present study. The corresponding increase in VLDL levels further supports this finding, as VLDL is the primary carrier of triglycerides. Karn et al., in their observational study, also reported significantly higher triglyceride levels in POAG patients compared to controls.[13] However, population-based and meta-analytic studies, including that by Yang et al., have demonstrated variability in the association between triglycerides and intraocular pressure, suggesting possible influence of demographic and metabolic factors.[10]

A significant reduction in HDL levels was observed in POAG patients in this study. HDL plays a protective role through its antioxidant, anti-inflammatory, and endothelial-stabilizing properties. Reduced HDL levels may increase susceptibility of the optic nerve head to ischemic damage. Joshi et al., in a hospital-based study, also reported significantly lower HDL levels in POAG patients.[12] Similarly, Pavljasević et al. observed reduced HDL levels in glaucoma patients compared to controls.[11]

LDL levels were significantly elevated in POAG patients in the present study. Elevated LDL is associated with atherosclerosis, endothelial dysfunction, and impaired microvascular circulation. Karn et al. demonstrated increased LDL levels in POAG patients in their clinical study.[13] In addition, large-scale epidemiological studies such as that by Ma et al., based on cohort data, have shown an association between serum lipid levels and glaucoma risk.[14]

The biological mechanisms linking dyslipidemia and POAG are likely multifactorial. Dyslipidemia can lead to endothelial dysfunction and impaired autoregulation of optic nerve head blood flow, resulting in reduced perfusion. Oxidative stress and lipid peroxidation may also damage trabecular meshwork cells, impair aqueous outflow, and increase susceptibility of retinal ganglion cells to apoptosis.[4,6]

Another important consideration is the role of systemic treatments such as statins. Observational studies have shown conflicting results regarding the protective or modifying role of statins in glaucoma. Xu et al., using a Mendelian randomization approach, suggested that the causal relationship between lipid levels and POAG remains uncertain.[15] Therefore, lipid-lowering therapy may act as a confounding factor in observational studies.

The findings of the present study are consistent with previous literature suggesting an association between dyslipidemia and POAG. However, as this is a case–control study, causality cannot be established.

From a clinical perspective, these results highlight the importance of evaluating systemic metabolic factors in patients with POAG. Lipid profile assessment may be useful as part of a comprehensive approach to glaucoma management.

CONCLUSION

The findings of the present study indicate that patients with primary open-angle glaucoma tend to exhibit an atherogenic lipid profile, characterized by elevated levels of total cholesterol, triglycerides, low-density lipoprotein (LDL), and very low-density lipoprotein (VLDL), along with reduced high-density lipoprotein (HDL) levels.

Dyslipidaemia may contribute to the pathogenesis of glaucomatous optic neuropathy through multiple mechanisms, including endothelial dysfunction, impaired autoregulation of optic nerve head perfusion, oxidative stress, and lipid peroxidation. These alterations may increase the susceptibility of retinal ganglion cells to damage and may also influence trabecular meshwork function, thereby affecting aqueous outflow.

However, as this is a case–control study, a causal relationship cannot be established. Further large-scale, longitudinal studies are required to better elucidate the role of lipid abnormalities in the development and progression of primary open-angle glaucoma.

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