Research Article

INTERNATIONAL RESEARCH JOURNAL OF PHARMACY

www.irjponline.com

ISSN 2230-8407 [LINKING]

A study of association of lipid profile with primary open angle glaucoma in non-obese patients

Dr Ekta Batavia[1], Dr Vikas Kumar Mishra[2], Dr Ravi Kant[3*]

[1]Assistant Professor, Department of Ophthalmology, Shri Rawatpura Sarkar Institute of Medical Sciences and Research, Raipur, CG, India
[2]Assistant Professor, Department of Ophthalmology, Shri Balaji Institute of Medical Sciences, Raipur, CG, India
[3]Associate Professor, Department of Biochemistry, SSIMS, Bhilai, CG, India.
Emai: rkks09@gmail.com

Corresponding Author
Email: rkks09@gmail.com

How to cite: Dr Ekta Batavia, Dr Vikas Kumar Mishra, Dr Ravi Kant. **A study of association of lipid profile with primary open angle glaucoma in non-obese patients.** International Research Journal of Pharmacy. 2025; 16:10: 08-14.

Doi: http://doi.org/10.56802/irjp.2025.v16.i10.pp08-14

==========

ABSTRACT

Background: Hyperlipidemia has been linked to glaucoma and elevated intraocular pressure (IOP).
Aim and Objective: To evaluate the association between serum lipid profile and Primary Open Angle Glaucoma (POAG) in non-obese patients.
Materials and Methods: This case–control study was conducted after approval from the Institutional Ethics Committee (IEC) and obtaining written informed consent. A total of 120 adults (>40 years) were enrolled: 60 non-obese POAG patients (cases) and 60 age-matched non-glaucoma subjects (controls). Serum lipid profiles were assessed using Vitros 5600, in accordance with the Declaration of Helsinki.
Results: Mean total cholesterol and triglyceride levels were significantly higher in non-obese POAG cases compared to controls ($p<0.05$), while mean high-density lipoprotein (HDL) levels were significantly lower ($p<0.001$). Elevated cholesterol levels were found to be significantly associated with POAG in non-obese patients ($p<0.001$).
Conclusion: Serum lipid abnormalities, particularly increased cholesterol and triglycerides with reduced HDL, are significantly associated with the risk of POAG in non-obese individuals.
Keywords: Glaucoma, Lipid Profile, Obase.

Introduction

The most common forms of glaucoma include open-angle glaucoma (OAG), normal-tension glaucoma (NTG), and angle-closure glaucoma (ACG). These subtypes can be further classified based on pathological causes as primary or secondary (e.g., trauma, disease, or exfoliation), by age of onset as adult, juvenile, or congenital forms, and by disease progression as chronic or acute. The diversity of subtypes makes glaucoma research challenging; therefore, many current studies focus on the most prevalent forms [1].

India is among the most affected countries, with over 12 million people suffering from Primary Open Angle Glaucoma (POAG) [2]. POAG is characterized by degeneration of retinal ganglion cells and subsequent optic nerve head changes. Its pathogenesis is multifactorial, involving elevated intraocular pressure (IOP), vascular insufficiency, oxidative stress, and immune mechanisms [3–4]. Hyperlipidemia has been documented to be associated with both POAG and increased IOP [4–8], with particular links to specific lipid fractions. To explore this further, we conducted a case–control study to evaluate the association of lipid profile with POAG in non-obese patients.

Materials and Methods

This case–control study was conducted in the Departments of Ophthalmology and Biochemistry at a tertiary care center after obtaining approval from the Institutional Ethics Committee (IEC) and written informed consent from all participants. The study adhered to the tenets of the Declaration of Helsinki.

Inclusion Criteria

A total of 120 patients aged above 50 years were enrolled and divided into two groups:

- **Cases:** 60 consecutive non-obese patients diagnosed with Primary Open Angle Glaucoma (POAG).
- **Controls:** 60 consecutive non-glaucoma patients.

Demographic data including age and gender were recorded. A general physical and systemic examination was conducted, and medical history was taken with particular attention to systemic illnesses (e.g., uncontrolled hypertension or uncontrolled diabetes mellitus [9–10]), drug intake, smoking, alcohol use, and family history of chronic diseases.

Exclusion Criteria

- Patients were excluded if they had any of the following:
- High myopia
- Uveitis
- Body Mass Index (BMI) >30.0
- Media opacities (corneal opacity, cataract)
- Retinal detachment, diabetic or hypertensive retinopathy, age-related macular edema

- Systemic conditions such as uncontrolled hypertension or uncontrolled diabetes mellitus [9–10]
- History of smoking or chronic alcoholism [11]

Ophthalmological Assessment

All patients underwent detailed ocular evaluation:

- **Visual acuity:** Best corrected visual acuity measured with Snellen's chart
- **Anterior and posterior segment evaluation:** Torch light test, pupillary reaction, anterior segment examination, slit-lamp examination, distant direct ophthalmoscopy
- **Intraocular pressure (IOP):** Measured with Goldmann applanation tonometer; anterior chamber depth assessed by Van Herick's grading.
- **Angle assessment:** Gonioscopy
- **Visual field analysis:** Standard automated perimetry using Humphrey Field Analyzer (HFA II 720-5545-3.2/3.2; Humphrey-Zeiss) with the SITA Standard Central 30-2 program.
- **Optic nerve and retinal imaging:** Cirrus HD Spectral Domain Optical Coherence Tomography (SD-OCT), direct ophthalmoscopy, and fundus photography using Carl Zeiss VISUCAM 524

Biochemical Analysis

Venous blood samples (5 ml) were collected under aseptic precautions from all participants. Serum lipid profiles were analyzed using Vitros 5600 autoanalyzer.

Triglycerides:

- Normal: <150 mg/dl
- Mild hypertriglyceridemia: 150–499 mg/dl
- Moderate: 500–886 mg/dl
- Severe: >886 mg/dl

HDL-C:

- Low: <40 mg/dl
- High: ≥60 mg/dl

Statistical Analysis

Data were analyzed using **SPSS software, version 24.0**. Results were expressed as numbers and percentages. Chi-square test, independent samples *t*-test, and ANOVA were applied where appropriate. A *p*-value <0.05 was considered statistically significant.

Results

Table 1: Comparison of lipid level between non-obese POAG and controls

Parameter	Non-Obese POAG (n=60) (mean±SD)	Controls (n=60) (mean±SD)	P-value
Total cholesterol (mg/dl)	204.8±20.5	187.05±21.55	0.000009
Total triglyceride (mg/dl)	234.8±37.3	183.9±35.9	0.000001
HDL (mg/dl)	34.8±2.6	59.03±11.01	0.000001

Table 2: Comparison of total cholesterol between non-obese POAG and controls

Parameter	Non-Obese POAG (n=60)		Controls (n=60)		P-value
	No.	%	No	%	
Normal cholesterol (<200mg/dl)	28	46.7	40	66.7	0.027
Borderline high Cholesterol (>200-239 mg/dl)	13	21.7	20	33.3	0.152
High cholesterol (>240mg/dl)	19	31.6	0	0	0.0001

Table 3: Comparison of triglyceride between non-obese POAG and controls

Parameter	Non-Obese POAG (n=60)		Controls (n=60)		P-value
	No.	%	No	%	
Normal TG (<150mg/dl)	15	25	25	41.7	0.523
Borderline high TG (>150-499 mg/dl)	45	75	35	58.3	0.0523
High TG (>880mg/dl)	0	0	0	0	0

Table 4: Comparison of HDL between non-obese POAG and controls

Parameter	Non-Obese POAG (n=60)		Controls (n=60)		P-value
	No.	%	No	%	
Normal HDL (<40mg/dl)	23	38.33	48	80	0.0001
Borderline high HDL (>40-60 mg/dl)	37	61.7	12	20	0.0001
High HDL (>60mg/dl)	0	0	0	0	0

Discussion

Glaucoma is a progressive optic neuropathy and remains one of the leading causes of irreversible blindness worldwide [8]. Lipid dysregulation has emerged as an important factor in its pathogenesis. Persistent dyslipidemia can induce degenerative changes in the retinal and choroidal vasculature, leading to ischemic retino-neural angiopathy [12–13], hypoperfusion, and disruption of ganglion cell microcirculation, ultimately resulting in permanent visual impairment [14]. The underlying mechanisms may involve oxidative stress and atherogenic changes triggered by altered lipid metabolism [15–16].

The present study is distinct in that it specifically examined the relationship between serum lipid levels and POAG in non-obese patients, assessing both mean values and categorical cut-offs. Our findings indicate that total cholesterol, triglyceride, and LDL levels were significantly higher in POAG cases compared to controls, while HDL levels were significantly lower. Importantly, higher total cholesterol, triglyceride, LDL, and lower HDL were associated with increased odds of POAG in non-obese patients.

The role of HDL-C in glaucoma remains controversial. While several studies report no significant association or even a protective effect, others suggest that higher HDL-C may be linked with elevated IOP and increased glaucoma risk [9,11,17–20]. Despite such variability, dyslipidemia is now widely recognized as a potential risk factor for glaucoma. Moreover, changes in the retinal vasculature, such as narrowing of arterioles and altered blood flow velocity, have been shown to contribute to glaucomatous damage [10].

Our results align with earlier studies. For example, one study reported mean serum total cholesterol and triglycerides significantly higher in POAG cases (211.2±51.9 and 165.9±88.6 mg/dl) compared to controls (162.3±39.6 and 99.5±43.1 mg/dl), with hypercholesterolemia and hypertriglyceridemia more frequent among cases [20]. Similarly, another case–control study found higher rates of dyslipidemia (hypercholesterolemia, hypertriglyceridemia, high LDL, and low HDL) in POAG patients than in controls, reinforcing the role of altered lipid metabolism in glaucoma development.

Gupta et al. [22] compared lipid levels between 100 POAG cases and 100 controls, finding significantly higher TC, TG, and LDL in cases, with dyslipidemia recognized as an independent risk factor. Roy and Ghanta [23] also reported significantly higher cholesterol, triglyceride, and LDL levels in POAG cases compared to controls. Our findings are consistent with these results and further demonstrate that even in non-obese adults, serum lipid abnormalities are strongly associated with POAG.

Conclusion

In this study, non-obese POAG patients exhibited significantly higher total cholesterol, triglyceride, and LDL levels, along with significantly lower HDL levels, compared to controls. These lipid abnormalities were strongly associated with increased odds of POAG. Our findings support the growing evidence that dyslipidemia is an important risk factor for glaucoma, independent of obesity. However, the study has limitations, including a relatively small sample size, open analytical approach, and the possibility

of incidental associations due to limited numbers. Larger, multicentric studies with more rigorous controls are warranted to validate these findings and clarify the precise role of serum lipids in glaucoma pathogenesis.

Source of Funding: Nil.

Reference

1. Quigley, H.A.; Broman, A.T. The Number of People with Glaucoma Worldwide in 2010 and 2020. *Br. J. Ophthalmol.* **2016**, *90*, 262–267.
2. Saxena R, Singh D, Vashist P. Glaucoma: an emerging peril. *Indian J Community Med*. 2013;38(3):135–7.
3. Agarwal R, Gupta SK, Agarwal P, Saxena R, Agrawal SS. Current concepts in the pathophysiology of glaucoma. *Indian J Ophthalmol*. 2009;57(4):257–66.
4. Rieck J. The pathogenesis of glaucoma in the interplay with the immune system. *Invest Ophthalmol Vis Sci*. 2013;54(3):2393–409.
5. Wang S, Bao X. Hyperlipidemia, blood lipid level, and the risk of glaucoma: A meta-analysis. *Invest Ophthalmol Vis Sci*. 2019;60(4):1028–43.
6. Dube M, Chhawania PK, Shukla A, Kujur R, Tiwari U. Correlation between serum lipids and primary open angle glaucoma: A clinical study. *Delhi J Ophthalmol*. 2019;29(4).
7. Lee Y, Siddiqui WJ. Cholesterol levels; 2023. Available from: https: //www.ncbi.nlm.nih.gov/books /NBK542294/.
8. Weinreb RN, Aung T, Medeiros FA. The pathophysiology and treatment of glaucoma: a review: A review. *JAMA*. 2014;311(18):1901–11.
9. Kim YJ, Chun YS, Lee MY, Kim JM, Shim SH, Yoo C, et al. Association of IOP with systemic factors in a Korean cohort. *Optom Vis Sci*. 2015;92(12):1182–8.
10. Modrzejewska M, Grzesiak W, Zaborski D, Modrzejewska A. The role of lipid dysregulation and vascular risk factors in glaucomatous retrobulbar circulation. *Bosn J Basic Med Sci*. 2015;15(2):50–6.
11. Yokomichi H, Kashiwagi K, Kitamura K, Yoda Y, Tsuji M, Mochizuki M, et al. Evaluation of the associations between changes in intraocular pressure and metabolic syndrome parameters: a retrospective cohort study in Japan. *BMJ Open*. 2016;6(3):010360.
12. Deokule S, Vizzeri G, Boehm AG, Bowd C, Medeiros FA, Weinreb RN. Correlation among choroidal, parapapillary, and retrobulbar vascular parameters in glaucoma. *Am J Ophthalmol*. 2009;147(4):736–43.
13. Wong TY, Klein R, Sharrett AR, Nieto FJ, Boland LL, Couper DJ, et al. Retinal microvascular abnormalities and cognitive impairment in middle-aged persons. The atherosclerosis risk in communities study. *Stroke*. 2002;33(6):1487–92.
14. Martínez A, Sánchez M. Predictive value of colour Doppler imaging in a prospective study of visual field progression in primary open-angle glaucoma: Acta Ophthalmologica Scandinavica. *Acta Ophthalmol Scand*. 2005;83(6):716–22.
15. Yang RL, Shi YH, Hao G, Li W, Le GW. Increasing oxidative stress with progressive hyperlipidemia in human: Relation between malondialdehyde and atherogenic index. *J Clin Biochem Nutr*. 2008;43(3):154–8.
16. Yilmaz N, Coban DT, Bayindir A, Erol MK, Ellidag HY, Giray O, et al. Higher serum lipids and oxidative stress in patients with normal tension glaucoma, but not pseudoexfoliative glaucoma. *Bosn J Basic Med Sci*. 2016;16(1):21–7.

17. Cui Y, Yang X, Zhang G, Guo H, Zhang M, Zhang L, et al. Intraocular pressure in general and diabetic populations from Southern China: The Dongguan Eye Study. *Invest Ophthalmol Vis Sci*. 2019;60(2):761–69.
18. Wang YX, Tao JX, Yao Y. The association of intraocular pressure with metabolic syndrome and its components: a Meta-analysis and systematic review. *Int J Ophthalmol*. 2019;12(3):510–16.
19. Shon K, Sung KR. Dyslipidemia, dyslipidemia treatment, and open-angle glaucoma in the Korean national health and Nutrition Examination Survey. *J Glaucoma*. 2019;28(6):550–6.
20. Kim MJ, Kim MJ, Kim HS, Jeoung JW, Park KH. Risk factors for open-angle glaucoma with normal baseline intraocular pressure in a young population: the Korea National Health and Nutrition Examination Survey: Risk factors for young adult glaucoma. *Clin Exp Ophthalmol*. 2014;42(9):825–32.
21. Agarwal R, Gupta SK, Agarwal P, Saxena R, Agrawal SS. Current concepts in the pathophysiology of glaucoma. *Indian J Ophthalmol*. 2009;57(4):257–66.
22. Gupta R, Sharma A, Sharma HR. Dyslipidemia in Primary Open Angle Glaucoma. *JK Sci*. 2020;22(2):84–7.
23. Roy S, Ghanta D. A clinical study showing association of serum lipids with primary open angle glaucoma. *Int J Sci Res*. 2021;10(3):63–4.