

A Clinical Study on the Correlation between Primary Open Angle Glaucoma and Serum Lipids

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Abstract:

Background: One of the leading causes of blindness in the world is Glaucoma. The primary risk factor is Intra Ocular Pressure. But some cases progress even with control of intra ocular pressure. This substantiates the view that there are independent risk factors in pathogenesis of Glaucoma. This study was carried out by us to study the relation between Primary Open Angle Glaucoma and Serum Lipids.

Methods and Materials: This study was conducted on 45 patients with POAG and 45 individuals without Glaucoma (Controls). Ophthalmic examination was performed on all patients and Fasting Lipid profile including Total Cholesterol, Low density lipoprotein, High Density Lipoprotein and Triglycerides were measured and analysed between cases and controls.

Results: It was noted that the level of total cholesterol, total glycerol and LDL were significantly higher in cases than in controls with P values < 0.06. level of HDL was lower in controls but it was not statistically significant.

Conclusion: Dyslipidemia is an independent risk factor for POAG. High levels of Serum Cholesterol, Serum Triglycerides and serum LDL correlate significantly with POAG.

Keywords: lipids, dyslipidemia, primary open angle glaucoma, correlation.

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Introduction

The second leading cause of blindness in India is primary open angle glaucoma (POAG) Which is a chronic progressive eye disease. There are around 11 million people in India aged 40 years and above with glaucoma. 6.49 million people are estimated to be affected by POAG. It usually manifests in the advanced stages of the disease. It is therefore necessary to identify its risk factors. In the incidence of this disease, different factors have a role to play. The primary risk factor is high intra ocular pressure (IOP). Raised IOP leads to

Optic Nerve damage either by direct mechanical damage to retinal nerve fibre layer or damage due to compression of blood vessels supplying optic nerve head. It is understood that there may be other risk factors also which by modulating the IOP or other different mechanisms, may lead to causation and progression of POAG.

Diabetes Mellitus (DM) Type II and Hypertension are known to be associated with POAG. Hypertension and Diabetes are linked to high lipid levels. Dyslipidemia

and Insulin resistance being interrelated and high lipid levels cause atherosclerotic changes leading to hypertension. Therefore there is a possibility that Glaucoma is inherently related to Serum Lipid levels.

Lipid peroxidation can lead to oxidative stress, which may directly damage trabecular meshwork and endothelium of blood vessels supplying optic nerve head or atherosclerotic changes due to elevated cholesterol levels which may affect ocular perfusion. The purpose of this study is to establish a relation between Serum lipid levels and primary open angle glaucoma.

Materials and Methods

This case control study was conducted in the Department of Ophthalmology in Akash Institute of Medical Sciences and Research Centre, Bangalore, Karnataka, from July 2022 to December 2022. In this study 45 patients of POAG and 45 healthy volunteers were included. Written informed consent was taken from all subjects. Primary open angle glaucoma patients over 18 years of age having no other ocular disease were included in the study. History of Ocular trauma systemic diseases like DM and Hypertension with ocular involvement were excluded. Demographic data including age, gender, address were recorded. Visual acuity with Snellen's chart, Anterior segment

examination with slit lamp, pupillary reaction, IOP measurement using Goldman Applanation tonometer. Angle structures were evaluated by Gonioscopy. Fundus examination was done and mild defects were recorded. POAG was diagnosed on the basis of raised IOP, ONH changes which was detected by direct Ophthalmoscopy and visual field defects.

Twelve hour fasting blood samples were collected for measuring serum lipids and assessed using enzymatic method (Auto analyzer). The lipid profile included Total cholesterol, Triglycerides (TGL), low density lipoproteins (LDL) and High Density lipoprotein (HDL). Reference values were taken from cholesterol evaluation program. Adult treatment panel iii guidelines according to which Hypercholesterolemia is defined as Total cholesterol >200 mg/dl. Hypertriglyceridemia when triglycerides >150 mg/dl, LDL >130 mg/dl were considered high and HDL 40 mg/dl were considered low.

Standard mean deviation and standard error of means were calculated. Statistical analysis was performed using unpaired T test using SPSS software. P value, 0.05 was considered significant.

Results

Table I: Demographic patterns of study population

Parameter	cases (n=45)	Controls (n=45)
Mean Age (years)	58 yrs.	56 yrs.
Gender (Male::Female)	14:8	24:18
Obesity (BMI * >-25)	8/45 (15%)	4/45 (5%)
Locality	32/45 rural (75%)	24/45 rural (55%)

*BMI – Body Mass Index

The age of patients aged from 35-75 years with mean age 54 years(cases) and 52 years(controls) The maximum number of cases and controls were between 45-55 years of age. Male to female ratio was 15:8 in cases and 24; 18 in controls.in out of 45(48.5%) cases were overweight, obese, 7 obese and 14 overweight. High cholesterol

(>200 mg/dl) was seen in 22 cases, in controls 8 had high cholesterol. High triglycerides (>150 mg/dl) was seen in control 8 cases. LDL was high (>130 mg/dl) in 25 cases and 9 controls. HDL was (<40 mg/dl) in 29 cases and 15 controls.

Table 2: Dyslipidemias in cases and controls

Lipid parameters	Cases (n=45)	controls (n=45)
High cholesterol (> 200mg/dl)	22/45 (49%)	9/45(18%)
High Triglycerides (>50mg/dl)	20/45 (45%)	8/45 (15%)
High LDL (>130mg/dl)	25/45 (60%)	9/45 (18%)
Low HDL (< 40mg/dl)	29/45 (68%)	15/45 (32%)

In cases, mean total cholesterol was 215.90± 6.42 mg/dl; mean triglycerides was 152.05 ± 7.80mg/dl ;mean LDL level was 140.7±6.20 mg/dl and mean HDL was 38.60±1.50 mg/dl; In controls mean total cholesterol was 160.45± 6.16 mg/dl; mean triglycerides was 109.60± 7.32mg/dl; mean LDL Level was 104.3 ± 5.49 and mean HDL was 41.93±1.68 mg/dl

Table 3: Serum Lipid values in Cases and Controls

Parameters	Cases(n=45)	Controls (n=45)
Mean Cholesterol (mg/dl)	215.90 ± 6.42	160.45± 6.16
Mean Triglycerides (mg/dl)	152.05 ± 7.80	109.60± 7.32
Mean LDL (mg/dl)	140.7 ± 6.20	104.3±5.49
Mean HDL (mg/dl)	38.60± 1.50	41.93± 1.68

Levels of total cholesterol, total triglycerides and LDL were significantly high in cases than in controls with P value < 0.05 confidence interval 95%. Level of HDL was lower in cases than in controls but it was not statistically significant.

Discussion

It is evident through results obtained that there is a significant relationship between high cholesterol, LDL, Triglycerides and low HDL to POAG. HDL was found to be lower in cases than controls, but this was not statistically significant.

It was concluded by Koveriak et al in their study that patients with higher cholesterol, particularly atherogenic LDL fraction may have certain influence on glaucoma. Serum lipid levels similar in both groups for triglycerides, HDL and LDL but cholesterol levels were increased in POAG group.

Egorov et al showed that lipid biochemical analysis in patients with glaucoma may have atherogenic hyperlipidemia with lower antioxidative activity. Statins may significantly lower the risk of glaucoma if used for longer than 24 months. The statins usage in hyperlipidemia therapy could not change IOP status in patients with glaucoma but could reduce risk of

glaucoma. In a case control study conducted by Davie et al there was partial relation between POAG and Dyslipidemia. OR= 7.15 (95 % CI :23-22.2) for hypercholesterolemia and OR= 16.9 (95% CI: 2.1 -14) For Hypertriglyceridemia. It was concluded that hyperlipidemia can be a risk factor for POAG.

Paveljasevic S and Aseric M conducted a similar study in Bosnia, the researchers tested 50 patients with open angle glaucoma and 50 healthy individuals with respect to their serum lipid levels. The mean cholesterol value in the test group was 6.14 mol/dm and in the control group it was 5.96 mol/dm. The triglyceride mean value in test group was 2.04 mol/mm. HDL was average in test group with 1.45 mol/dm and in test group 1.40 mol/mm. LDL was 3.98 mol/mm in test group and in control 4.0 mol.mm. We can incur that blood cholesterol levels for patients in the test group were higher compared to those of the control group and suggests that hypercholesterolemia could be a predictable factor in POAG diagnosis.

In Beijing eye study, 3251 individuals (>45yrs) had complete ophthalmic examination, serum lipids were measured. After adjustment of various factors such as

age, sex, income, BMI, serology, blood sugar, blood pressure, the effect of dyslipidemia on incidence of ophthalmic diseases were studied. The results showed that IOP was significantly raised in dyslipidemic patients.

A study done by Chivastum and Strial in 1988 on 183 patients (92 women and 91 men) with glaucoma with aim of surveying serum lipid levels it was found that only triglycerides in females was significantly high. Stewart et al conducted a study in 1996 and made a comparison between total cholesterol and HDL of 25 glaucoma patients and healthy individuals which showed that there is no relationship between HDL and total cholesterol to IOP or POAG.

The relationship between lipids and glaucoma could be due to association of these diseases with other cardiac risk factors such as diabetes and hypertension. In a cohort study conducted in Michigan university, people aged > 40 years who had one or more ophthalmic visit, elements of metabolic syndrome and glaucoma were examined, It was found through the results that DM and HTN are associated with each other and have a role in occurrence of glaucoma but dyslipidemia alone lowered risk of glaucoma by 5%. But in cases that dyslipidemia is associated with Hypertension or Diabetes Mellitus the risk increases in pathogenesis of glaucoma. It was found in another study that dyslipidemia is an independent risk factor for glaucoma after removing these confounding factors.

These studies help in understanding the mechanisms by which dyslipidemia leads to development of POAG. Many studies showing increased levels of lipid peroxides in the aqueous humor, trabecular meshwork and Schlemm's canal in POAG cases. This suggests that lipid peroxidation by increasing oxidative stress is responsible for destruction of the trabecular meshwork and Schlemm's canal.

Conclusion: we can conclude that Dyslipidemia is an independent risk factor for POAG. High serum cholesterol, high serum triglycerides and high serum LDL correlate significantly with POAG. Hence treatment of dyslipidemia can provide a potential preventing strategy for POAG.

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