Original Research Article

Exploring Lipid Abnormalities in Senile Cataract: A Cross-Sectional Investigation

Abstract

Background: Age-related cataracts (ARC) are a common cause of vision impairment in older adults. Previous studies have suggested that abnormal Lipid profile and oxidative stress may contribute to the development and progression of ARC.

Aim: The aim of this study was to compare the serum Lipid profile levels in patients with ARC and healthy controls.

Materials and Methods: This was a case-control study involving 50 patients with ARC and 50 age and gender-matched controls. Serum Lipid profile levels were measured using standard methods. The data were analyzed using t-test. A P-value of less than 0.05 was considered statistically significant.

Results: The results showed that the mean serum level of HDL Cholesterol was significantly lower in the ARC group than in the control group, while the mean serum level of triglyceride was significantly higher in the ARC group than in the control group.

Conclusion: This study indicates that altered lipid profile may be a risk factor for ARC.

Keywords:age-related cataract, serum lipid, case-control study, low-density lipoprotein cholesterol.

INTRODUCTION

Cataract is a major cause of blindness and low vision worldwide. It is estimated that 44.1% of blind cases and 51.6% of patients with low [2,3] vision suffers from cataract. Age is the most important risk factor and about 85 percent of involved patients have age-related cataract. This type of cataract is called "senile cataract". It has been shown to be the 4 main cause of blindness in patients over 50 years of age. In India cataract has been reported to be responsible for 50-80% of the bilateral [5,6] blindness in general. It is presumed that population above 60 years of [5,7] age which was around 56 million in 1991 may rise twice by 2016. It is estimated that a ten-year delay in the onset of cataracts could decrease the number of cataract surgeries by 45 per cent, thus considerably

Diminishing cost of care. Also, Patients with cataracts in low resource areas and developing countries have a poor chance for surgery due to economic reasons. This results in an increased risk of blindness in such [2,8] population. The development of senile cataract is a complex multifactorial process. Several factors such as genes, gender, diabetes, geographic location, UV light exposure, level of education, occupational status, nutritional factors and raised Body mass index [2]have been found to be associated with cataract formation. Some risk factors for cataract are modifiable; and the disease can be prevented by the elimination of these factors. It has recently been shown that dyslipidemic patients may develop lens opacities more frequently than the normal population, and thus lens opacities should be regarded as [9,10] one of the most common clinical signs of dyslipidemia. Also, some studies have been carried out relating components of metabolic syndrome with development of senile cataract which have shown dyslipidemia as risk factor for the development of age related cataract.[11]

The aim of this study was to investigate the biochemical changes in senile cataract patients.

MATERIAL AND METHODS

The study was carried out in the Department of Biochemistry, in collaboration with the Ophthalmology Department, from May 2021 to October 2022 in a Topiwala National Medical College Mumbai. The study sample included 120 participants aged 50 to 80 years, who were divided into two groups: cases and controls. Cases were patients diagnosed with senile cataract, and controls were normal healthyindividuals.

The inclusion criteria for the study were:

1) Diagnosis of senile cataract by an ophthalmologist

2) Normal healthy individuals without any eye diseases as controls

3) Age range of 50 to 80 years.

The exclusion criteria for the study were:

1) Refusal to participate in the study

2) Cataract caused by other factors such as trauma, metabolic disorders, radiation therapy etc.

3) Any systemic disease such as diabetes, hypertension etc.

4) Acute or chronic diarrhea

5) Acute or chronic renal failure

6) History of drug intake such as steroids, antipsychotics, chemotherapy etc.

We obtained 5 ml of blood from the veins of each participant after they had fasted for 10-12 hours overnight. We then separated the serum by spinning it in a machine and measured the levels of Lipid profile, which includes serum Total cholesterol, Triglyceride, HDL, and LDL, using a Beckman coulter Clinical chemistry Autoanalyzer.

We used Friedewald's equation to estimate serum VLDL levels. We obtained reagent kits from commercial sources and followed the manufacturer's instructions. Statistical analysis

We performed unpaired t-tests to compare the parameters between the case and control groups. We considered p-values less than 0.05 as statistically significant.

RESULTS

Table1: Comparison Of Lipid Profile In Senile Cataract Cases And Controls

Parameter	Cases (n=50) [mean ± SD]	Controls (n=50) [mean ± SD]	CI 95 %	P value
Age (year)	66.99±8.83	66.00±7.10	(0.19, 1.79)	0.48
BMI (kg/m^2)	23.68±3.16	21.55±2.74	(1.27, 2.99)	0.05
Smoking (yes/no)	35/15	22/38		
Triglycerides (mg/dL)	221.8 ± 19.99	139.5 ± 21.57	(62.4,102.2)	<0.0001
Total Cholesterol (mg/dL	173.10 ± 13.15	170.42 ± 20.22	(-8.76, 14.72)	0.2129
HDL-C (mg/dL)	38.3 ± 3.75	44.08 ± 6.79	(-9.45, -2.11)	<0.0001
LDL-C (mg/Ll)	108.44 ± 11.53	109.44 ± 12.52	(-5.67, 3.67)	0.834
VLDL-C (mg/dL)	41.36 ± 4.50	27.78 ± 4.47	(9.51, 17.65)	0.002

The table shows the comparison of some parameters between cases and controls in a study. The cases are patients of cataracts and the controls are healthy individuals. The parameters include age, body mass index (BMI), smoking status, and lipid profile. The table reports the mean and standard deviation (SD) of each parameter for both groups, as well as the 95% confidence interval (CI) and the p value of the difference between the groups. The p value indicates the statistical significance of the difference, with a lower value meaning a higher likelihood that the difference is not due to chance. The table reveals that there is no significant difference in age and total cholesterol between the cases and controls, as the p values are higher than 0.05. However, there is a significant difference in BMI, triglycerides, HDL-C, and VLDL-C between the groups, as the p values are lower than 0.05. The cases have higher BMI, triglycerides, and VLDL-C, and lower HDL-C than the controls.

DISCUSSION:

The results of this study showed that serum triglyceride and VLDL levels were significantly higher in senile cataract patients than in the control group ($p< 0.001$). This is in agreement with previous studies by Heydari B. et al (2012) and Hiller R. et al (2003) that also reported elevated serum triglyceride levels in senile cataract cases. Moreover, this study found that serum HDL levels were significantly lower in senile cataract patients than in the control group ($p< 0.001$), which is consistent with the findings of Heydari B et al (2012), Hiller R. et al (2003) and Meyer D. et al (2003).

One possible explanation for this is that increased BMI leads to insulin resistance mediated by adipokines and free fatty acids (FFA) even in the absence of clinical diabetes. Adipokines such as resistin and retinol-binding protein 4 reduce insulin sensitivity. Furthermore, cytokines like TNF-α and IL-6, which are produced by macrophages in adipose tissue, also play a role. Insulin resistance may cause hypertriglyceridemia due to increased free fatty acid influx to the liver, which results in hepatic accumulation of triglycerides. This leads to an increased hepatic production of very low density lipoproteins. This also interferes with the lipolysis of chylomicrons due to competition mainly at the level of lipoprotein lipase (LPL) with increased remnant triglyceride being transported to the liver. Lipolysis is further impaired in obesity by reduced mRNA expression of lipoprotein lipase in adipose tissue and reduced LPL activity in skeletal muscle.

Hypertriglyceridemia affects the distribution of cholesterol esters and triglycerides among VLDL, HDL and LDL through the action of cholesterylester transfer protein (CETP). This results in lower HDL-C levels and less TG content in LDL. The main pathophysiological mechanism that causes hypertriglyceridemia in senile cataract is the accumulation of triglyceride

rich lipoproteins such as VLDL, IDL, chylomicrons and their remnants, which may be due to increased synthesis (stimulated by insulin resistance) or decreased catabolism.

HDL has anti-inflammatory and antioxidant properties that help prevent the oxidation of LDL, a harmful type of cholesterol. HDL carries an enzyme called PON1, which can break down various compounds that are not normally found in the body, such as aryl esters. PON1 also protects LDL from oxidative damage. HDL and PON1 have a mutual relationship: HDL helps PON1 to be produced and function properly, while PON1 helps HDL to stay intact. PON1 activity can be reduced by lipid peroxidation, a process that damages cell membranes.

Jaouad et al. showed that PON1 activity is reduced by oxygen free radicals. Similarly, Hashim Z. et al reported lower Paraoxonase1 activity in senile cataract patients. They suggested that oxidative stress might cause the decline of PON1 activity in the plasma of these subjects, which could impair their ability to prevent lipid peroxidation. Therefore, low HDL level and high triglyceride level might be associated with lens opacities. However, more studies are needed to understand the exact mechanism of this association, as the literature is scarce. In our study, we did not find any significant difference in the serum levels of total cholesterol and LDL cholesterol between cases and controls. ($p > 0.05$). This result is in agreement with the previous studies by Hiller R. et al and Meyer D. et al.

CONCLUSION

Serum Triglyceride and VLDL levels were higher in patients with senile cataract than in healthy controls, while serum HDL-Cholesterol level was lower in patients with senile cataract than in healthy controls. These findings suggest that dyslipidemia may play a role in the onset and progression of senile cataract. Therefore, it is recommended that people maintain a normal BMI,

eat a balanced diet and enhance their antioxidant system by consuming vitamins such as tochopherols, ascorbic acid and retinoids, which may help prevent senile cataract.

REFERENCES

1. Khurana AK, Diseases of the Lens. Comprehensive Ophthalmology. 4th edition. New Delhi: New Age International (p) Limited; 2007. p.170

2. Heydari B, Kazemi T, Zarban A, Ghahramani S. Correlation of cataract with serum lipids, glucose and antioxidant activities: a case-control study. West Indian Med J. 2012; 61(3):230-4.

3. Resnikoff S, Pascolini D, Etya'ale D, Kocur I, Pararajasegaram R, Pokharel GP. Global data on visual impairment in the year 2002. Bull World Health Organ 2004; 82:844–51.

4. Hyman L. Epidemiology of eye disease in elderly. Eye. 1987; 1: 330-41.

5. Murthy G, Gupta S, John N, Vashist P. Current status of cataract blindness and Vision 2020: The right to sight initiative in India. Indian J Ophthalmol. 2008 Nov-Dec; 56(6):489–494.

6. Nirmalan P, Thulasiraj R, Maneksha V, Rahmathullah R, Ramakrishnan R, Padmavathi A. A population based eye survey of older adults in Tirunelveli district of south India: Blindness, cataract surgery and visual outcomes. Br J Ophthalmol. 2002; 86:505–12.

7. Kumar S. Alarm sounded over ′Greying′ of India′s population. Lancet. 1997;350:271.

8. Jose R, Bachani D. Performance of cataract surgery between April 2002 and March 2003. NPCB-India. 2003; 2:2.

9. Meyer D, Parkin DP, Maritz FJ, Liebenberg PH. Abnormal serum lipoprotein levels as a risk factor for the development of human lenticular opacities. Cardiovasc J South Afr. 2003; 14:60-64.

10. Meyer D, Parkin DP, Maritz FJ, Liebenberg PH. Cortical opacities in young patients- an indication for lipogram. S Afr Med J. 2001; 91:520-24.

11. Paunksnis A, BojarskieneF,Cimbalas A, Cerniauskiene L, Luksiene D. Relation between cataract and metabolic syndrome and its components. European Journal of Ophthalmology. 2007; 17(4):605-614.

12. Hiller R, Sperduto RD, Reed GF, D'Agostino RB, Wilson PW, et al. Serum lipids and agerelated lens opacities: a longitudinal investigation: the Framingham Studies. Ophthalmology. 2003 Mar; 110(3):578-83.

13. Klop B, Elte JWF, Cabezas MC. Dyslipidemia in obesity: mechanisms and p o t e n t i a l targets. Nutrients. 2013; 5:1218-1240.

14. Flock MR, Green MH, Kris-Etherton PM. Effects of adiposity on plasma lipid response to reductions in dietary saturated fatty acids and cholesterol. Adv. Nutr. 2011, 2, 261–274.

15. Murphy A, Chin-Dusting J, Sviridov D, Woollard K. The anti infammatory effects of high density lipoproteins. Curr Med Chem. 2009;16:667–75.

16. Efrat M, Aviram M. Paraoxonase 1 Interactions with HDL, Antioxidants a n d Macrophages Regulate Atherogenesis A Protective Role for HDL Phospholipids. Adv Exp Med Biol. 2010:153–66.

17. Tomas M, Latorre G, Senti M, Marrugat J. The Antioxidant Function of High Density Lipoproteins: A New Paradigm in Atherosclerosis.Rev Esp Cardiol. 2004; 57(6):557-69

18. Mackness B, Hine D, Liu Y, Mastorikou M, Mackness M. Paraoxonase-1 inhibits oxidised LDL-induced MCP-1 production by endothelial cells. BiochemBiophys Res Commun. 2004;318:680–683.

19. Deakin S, Leviev I, Gomaraschi M, Calabresi L, Franceschini G, James R W . Enzymatically active paraoxonase-1 is located at the external membrane of producing cells and released by a high af□nity, saturable, desorption mechanism. J Biol Chem. 2002; 277:4301–8.

20. James RW, Deakin SP. The importance of high-density lipoproteins for paraoxonase-1 secretion, stability, and activity. Free Radic Biol Med. 2004; 37:1986–94.

21. Wang M, Lang X, Cui S, Zou L, Cao J, Wang S, et al. Quantitative assessment of the influence of paraoxonase 1 activity and coronary heart disease risk. DNA Cell Biol. 2012; 31:975–82.

22. Hashim Z, Zarina S. Assessment of paraoxonase activity and lipid peroxidation levels in diabetic and senile subjects suffering from cataract. Clin. Biochem. 2007 Jun;40(9- 10):705–9.

23. Aviram M, Rosenblat M, Billecke S, Erogul J, Sorenson R, Bisgaier CL, et al. Human serum paraoxonase (PON 1) is inactivated by oxidized low density lipoprotein and preserved by antioxidants. Free Radic Biol Med 1999; 26:892–904.

24. Jaouad L, de Guise C, Berrougui H, Cloutier M, Isabelle M, Fulop Jr T, et al. Age-related decrease of high-density lipoproteins antioxidant activity is due to an alteration in the PON1's free sulfhydryl groups. Atherosclerosis 2006;185:191–200