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STUDY OF SERUM LIPID PROFILE IN PATEINTS OF SENILE CATARCT AND CONTROL GROUP.

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ABSTRACT:

BACKGROUND: Recent reports suggest an association between deranged Lipid profile, oxidative stress and age-related cataracts (ARC). Aim: This study was planned and carried out to study the Lipid profile level in Senile cataract patients. **MATERIALS AND METHODS:** We compared serum Lipid profile in 120 cases of ARC and 120 age and gender-matched controls. Statistical analysis was done using t-test. Significance was set at $P \leq 0.05$. **RESULTS:** The overall mean serum levels of HDL Cholesterol was significantly lower in the cases as compared to control group and serum triglyceride level was significantly higher in the cases as compared to control group. **CONCLUSION:** This study suggests that deranged lipid profile can be a risk factor for development and progression of senile cataract.

INTRODUCTION

Cataract is a major cause of blindness and low vision worldwide.[1] It is estimated that 44.1% of blind cases and 51.6% of patients with low vision suffers from cataract.[2,3] 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 main cause of blindness in patients over 50 years of age.[4] In India cataract has been reported to be responsible for 50-80% of the bilateral blindness in general.[5,6] It is presumed that population above 60 years of age which was around 56 million in 1991 may rise twice by 2016.[5,7] 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 population.[2,8] 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 have been found to be associated with cataract formation.[2] 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 one of the most common clinical signs of dyslipidemia.[9,10] 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]

MATERIAL AND METHODS

This study was conducted in the Department of Biochemistry, with the help of Ophthalmology Department during the period of May 2019 to October 2019 in tertiary health care institute. The study population consisted of total 240 participants aged between 50 to 80 years and they were divided in two groups viz cases and controls. Cases consisted of patients suffering from senile cataract and controls consisted of normal healthy individuals.

Inclusion criteria:1) Patients diagnosed as a case of senile cataract 2) Normal healthy individuals as control group 3) Age group of 50 to 80 years.

Exclusion criteria: 1) Not willing to participate in study 2) Cataract due to any other etiology like trauma, metabolic diseases, radiation therapy etc. 3) Any systemic disease like diabetes, hypertension etc. 4) Acute or chronic diarrhea 5) Patients of acute or chronic renal failure 6) Any H/O drug intake like steroid, antipsychotic, chemotherapy etc.

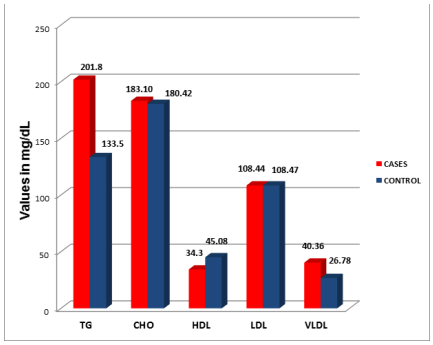
5 ml venous blood samples were collected after an overnight fast (10-12 hours) from all cases and controls and serum was separated by centrifugation and was analyzed for Lipid profile i.e, serum Total cholesterol, Triglyceride, HDL, LDL on Beckman coulter AU5800 Clinical chemistry Autoanalyzer. Serum VLDL was calculated by Friedewald's equation. Investigations were carried out using commercially available ready to use reagent kits. Statistical analysis Comparison of parameters was done between case and control group by using unpaired t- test. Interpretation was done according to p-value.

RESULT

TABLE: COMPARISON OF LIPID PROFILE IN SENILE CATARACT CASES AND CONTROLS

Parameter	Cases (n=120) [mean ± SD]	Controls (n=120) [mean ± SD]	P value
Triglycerides (mg/dL)	201.8 ± 20.99	133.5 ± 22.47	<0.0001
Total Cholesterol (mg/dL)	183.10 ± 12.05	180.42 ± 20.22	0.2129
HDL-C (mg/dL)	34.3 ± 3.35	45.08 ± 7.99	<0.0001
LDL-C (mg/Ll)	108.44 ± 12.52	108.47 ± 20.05	0.9895
VLDL-C (mg/dL)	40.36 ± 4.20	26.78 ± 4.47	<0.0001

GRAPH: COMPARISON OF MEAN SERUM LIPID PROFILE IN SENILE CATARACT CASES AND CONTROLS



DISCUSSION:

In present study it was found that serum triglyceride and VLDL level were found to be more significantly elevated in senile cataract cases as compared to control group ($p < 0.001$). Our study was consistent with the findings of Heydari B. et al (2012)[2], Hiller R. et al (2003)[12] with respect to serum triglyceride. In their studies they found higher serum triglyceride level in senile cataract patients as compared to control group.

Also, in our study serum HDL was more significantly decreased in senile cataract cases as compared to the control group ($p < 0.001$) in our study which was consistent with findings of Heydari B et al (2012)[2], Hiller R. et al (2003)[12] and Meyer D. et al (2003).[9]

The reason behind this could be that the raised BMI leads to development of 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 decrease insulin sensitivity. In addition, cytokines like TNF-α and IL-6, which originate from macrophages in adipose tissue, are also involved.[13,14] Insulin resistance may lead to hypertriglyceridemia which is due to increased free fatty acid influx to the liver, which leads to hepatic accumulation of triglycerides. This leads to an increased hepatic synthesis of very low density lipoproteins. This in turn hampers 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.[13]

Hypertriglyceridemia further induces an increased exchange of cholesterol esters and triglycerides between VLDL, HDL and LDL by cholesterylester transfer protein (CETP). This leads to decreased HDL-C concentrations and a reduction in TG content in LDL.[13]

So, the most important pathophysiological mechanism underlying the development of hypertriglyceridemia in senile cataract was accumulation of triglyceride rich lipoproteins i.e. VLDL, IDL, chylomicrons and their remnants might be due to increased synthesis (promoted by insulin resistance) or decreased catabolism. Also, one of the best known anti-inflammatory and antioxidant functions of HDL is its ability to inhibit the oxidation of low-density lipoprotein.[131] HDL exerts its inhibitory effects on oxidative modification of LDL by its related enzyme paraoxonase-1 (PON1) which can hydrolyze lactones, and several non-physiological substrates, such as aryl esters.[16] PON1 is transported in plasma as a component of HDL, and many studies have also shown that PON1 inhibits LDL oxidation.[17,18]

There is a close physiological association between PON1 activity and HDL in plasma. HDL facilitates the secretion of the PON1 by the liver and also stabilizes the enzyme,[19] and provides a hydrophobic environment which is needed for PON1 function.[20] In return, PON1 prevents the oxidation of HDL.[21] PON1 activity is known to be inhibited by lipid peroxidation.[22,23]

Recently, Jaouad et al. (2006)[24] demonstrated that exposure of PON1 to oxygen free radicals induces a significant decrease in PON1 activity. Also, Hashim Z. et al (2007)[22] documented decreased Paraoxonase1 activity in patients of senile cataract. The observed decline in plasma PON1 activity among senile subjects suffering from cataract appears to be mainly a consequence of oxidative stress.[22] So, decreased HDL level may be associated with reduced Paraoxonase1 activity and this may lead to insufficiency to protect against lipid peroxidation in general.

However, at this junction further extensive studies are required to know that how exactly increased triglyceride and decreased HDL levels leads to lens opacities, as there are very few reports available.

In our study, the comparison between the serum levels of total cholesterol and LDL cholesterol in cases and control was not statistically significant. ($p > 0.05$). This finding was consistent with the previous studies done by Hiller R. et al (2003)[12] and Meyer D. et al (2003).[9]

CONCLUSION:

Serum Triglyceride and VLDL level were significantly raised in senile cataract cases compared to controls and serum HDL-Cholesterol level was significantly low in senile cataract cases compared to controls. So, it can be concluded that dyslipidemia can be associated with development and progression of senile catarct. Hence, maintaining normal BMI, healthy diet & strengthening antioxidant system by consuming antioxidative vitamins like tochopherols, ascorbic acid and retinoids may be beneficial to prevent development of senile cataract.

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