



Lipid profile among asymptomatic individuals with history of DM/HTN

Dr. Mohammed Asif Ali, Dr. Prashanth ED

Assistant Professor, General medicine, Khaja Banda Nawaz institute of medical sciences, Kalaburagi, Karnataka, India

Abstract

Introduction: Lipoproteins are globular particles with a hydrophobic oily core consisting of nonpolar lipids (triglycerol and cholesterol esters) coated with native surfactants (phospholipids and free cholesterol) and specific proteins called apolipoproteins. These apo-protein decide the physiological role of a lipoprotein like binding to specific enzyme or to cell membrane, thus directing the lipoprotein to its sites of metabolism

Methodology: A detailed history and clinical examination was done in all patients. Blood samples were taken from all patients after a minimum of 12 hours of fasting, About 8ml of venous blood was drawn from the patients. 2ml of blood was collected in oxalate fluoride tube for blood glucose and 6ml was collected in dried glass tube for estimation of TC, TG, HDL. After clot retraction occurred the serum was separated and centrifuged at the rate of five thousand revolutions per minute for about five minutes

Results: The levels of various lipids (mean \pm SD) in the overall study population were total cholesterol 163 \pm 24, Triglycerides 115 \pm 34, LDL cholesterol 88 \pm 23 and HDL cholesterol 51 \pm 5. Age group classification of these lipid values showed that there was a significant increase in triglycerides (F=7.841; P<0.0001) with increasing age, HDL cholesterol showed a significant decrease (F=2.801; P <0.026) with increasing age. Total cholesterol levels were higher in older (41-45 years) as compared to younger subjects.

Conclusion: Though LDL cholesterol were higher in younger (20-25 years) age group a rising trend with age was noted.

Keywords: lipid profile, hypertension, diabetes

Introduction

Lipids are the organic substances insoluble in water, soluble in organic solvents, forms important dietary constituents with high caloric value, sources of fat soluble vitamins and essential fatty acids.

The major classes of plasma lipids are cholesterol, cholesteryl ester, triglyceride and phospholipid. Lipids are vital components of many of the body tissues. They are insoluble in water. To reach those tissues, lipids must be transported in the blood stream by complex, water soluble molecules called lipoproteins^[1, 2].

Lipoproteins are globular particles with a hydrophobic oily core consisting of nonpolar lipids (triglycerol and cholesterol esters) coated with native surfactants (phospholipids and free cholesterol) and specific proteins called apolipoproteins. These apo-protein decide the physiological role of a lipoprotein like binding to specific enzyme or to cell membrane, thus directing the lipoprotein to its sites of metabolism.

The term 'Atherogenic Dyslipidemia' denotes a combination of elevated triglycerides and small-dense LDL particles, and low levels of HDL-cholesterol, Dyslipidemia, particularly hypercholesterolemia and atherogenic dyslipidemia, have been closely implicated in the pathogenesis of CHD^[3].

The induction of hypercholesterolemia is a prerequisite for atherogenesis, studies across different populations reveal that those with higher cholesterol levels have more atherosclerosis and CHD than do those having lower levels. Epidemiological investigations of human populations incriminate high levels of

LDL cholesterol as being atherogenic. In population studies, the serum total cholesterol is a good surrogate for LDL-cholesterol levels. The Framingham Heart study. The multiple risk factor intervention trial (mrfit), and The Lipid Research Clinics (LRC) trial found a direct relation between levels of LDL cholesterol and the rate of new-onset CHD in men and women who were initially free of CHD.

Atherosclerosis generally can first be identified by gross pathological examination of coronary arteries in adolescence or early adulthood. The subsequent rate of atherogenesis is proportional to the severity of ambient risk factors including serum cholesterol levels. Moreover, the cholesterol level in young adulthood predicts development of CHD later in life. In three prospective studies with long-term followup, detection of elevated serum cholesterol in early adulthood predicted an increased incidence of CHD in middle-age^[4].

The relation of elevated LDL cholesterol to the development of CHD must be viewed as a multi-step process beginning relatively early in life.

Elevated serum triglycerides as a risk factor for CHD has been stimulated by the publication of meta-analysis that found that raised triglycerides are in fact an independent risk factor for CHD^[5].

The lipoprotein remnants include small very low density lipoproteins (VLDL) and intermediate density lipoproteins (IDL). They are cholesterol enriched particles and have many of the properties of LDL. In several clinical studies in which remnants were specifically identified, their elevations emerged as a strong predictors of coronary atherosclerosis or CHD^[2, 4].

Strong epidemiological evidence links low levels of serum LDL cholesterol to increased CHD morbidity and mortality. High HDL-cholesterol levels conversely convey reduced risk.

Methodology

A total of 274 young asymptomatic adults were selected on the basis of stratified random sampling.

A detailed history and clinical examination was done in all patients. Blood samples were taken from all patients after a minimum of 12 hours of fasting. About 8ml of venous blood was drawn from the patients. 2ml of blood was collected in oxalate fluoride tube for blood glucose and 6ml was collected in dried glass tube for estimation of TC, TG, HDL. After clot retraction occurred the serum was separated and centrifuged at the rate of five thousand revolutions per minute for about five minutes. The samples were analysed on the same day or within 48 hours by commercially available standard enzymatic kits (Accurex kits) for TC, TG and HDL-C, LDL-C levels were calculated using Friedwald's formula and dyslipidemias diagnosed as per NCEP guidelines.

Triglyceride estimation was done using Autozyme NEW Triglycerides reagent, based on enzymatic method using Lipoprotein Lipase glycerol Kinase, Glycerol Phosphate oxidase and peroxidase. Results were analysed on a Automated analyzer.

Statistical Methods Applied

Following statistical methods were employed in the present study

- Frequencies/ Descriptives
- Contingency coefficient (Cross Tabs)
- Chi-square test
- One-way ANOVA

Results

Table 1: Lipid concentrations (mg/dl) in various age groups

Age	TC	TG	HDL	LDL
	Mean±SD	Mean±SD	Mean±SD	Mean±SD
20-25	164±22	111±25	51±4.0	90±21
26-30	159±21	107±15	52±3.0	85±22
31-35	165±17	118±22	50±5	86±22
36-40	165±31	124±54	51±6	88±26
41-45	170±29	145±60	48±7	87±30
Total	163±24	115±33	51±4	88±23

TC- F=1.336, P<0.257; TG – F=7.841, P<0.0001; HDL-C-F=2.801, P<0.026; LDL-C- F=0.554, P<0.696

The levels of various lipids (mean± SD) in the overall study population were total cholesterol 163±24, Triglycerides 115±34, LDL cholesterol 88±23 and HDL cholesterol 51±5. Age group classification of these lipid values showed that there was a significant increase in triglycerides (F=7.841; P<0.0001) with increasing age, HDL cholesterol showed a significant decrease (F =2.801; P <0.026) with increasing age. Total cholesterol levels were higher in older (41-45 years) as compared to younger subjects. Though LDL cholesterol were higher in younger (20-25 years) age group a rising trend with age was noted.

Table 2: Lipid concentrations (mg/dl) in different study groups

Family history of	TC	TG	HDL	LDL
	Mean±SD	Mean±SD	Mean±SD	Mean±SD
HTN+	162±17	113±34	52±4	88±15
HTN-	155±24	111±29	51±5	82±21
DM+	162±22	113±22	51±4	89±20
DM-	168±22	114±27	52±3	92±21
HTN, DM+	167±35	126±55	49±6	92±32
HTN, DM-	164±20	114±28	52±4	86±24
Total	163±24	115±33	51±4	88±23

TC- F=1.661, P<0.144; TG – F=1.015, P<0.409; HDL-C- F=2.034, P<0.074; LDL-C- F=1.157, P<0.331

There was no statistical significant difference in lipid concentrations when groups are compared. Total cholesterol was more in individuals with family history of diabetic mellitus, triglycerides and LDL cholesterol were higher in individuals, with family history of DM and HTN, HDL cholesterol was lower in subjects with family history of DM and HTN.

Discussion

The present study is a hospital based descriptive study of 274 young asymptomatic individuals between 20-45 years, all the subjects were evaluated to detect the presence of dyslipidemia with special reference to family history of diabetes mellitus and hypertension.

The incidence of dyslipidemia in the study was 12.4% (34 out of 274 subjects showed lipid abnormalities). The most common lipid abnormality noted was hypertriglyceridemia (47.0%), next common lipid abnormality was low levels of HDL cholesterol (26.4%).

Though there was no statistical significant difference in lipid levels in different groups, the mean levels of total cholesterol, LDL cholesterol and triglycerides were more and HDL cholesterol was low in subjects with family history of diabetes mellitus and hypertension as compared to other groups.

The mean levels of total cholesterol, triglycerides and LDL cholesterol were lower in individuals without family history of hypertension as compared to other groups.

Combined dyslipidemias were more common in subjects belonging to group with family history of diabetes and hypertension.

In the present study the mean levels of lipids were as follows, total cholesterol 163 ±24, triglycerides 115±33; HDL 51±5 and LDL was 88 ± 23.

The results were consistent with other studies [6-9].

Conclusion

- The values of all the lipids like total cholesterol, triglycerides and LDL cholesterol were comparatively more in individuals with family history of diabetes mellitus and hypertension.
- HDL cholesterol was towards lower side in individuals with family history of diabetes mellitus and hypertension.

References

1. Larsen, Kronenberg, Melmed, Polonsky. William text

- book of endocrinology. 10th edition. Philadelphia: Saunders, 2003.
2. Donald B. Hunninghake. The medical clinics of North America – Lipid disorders. Philadelphia: W.B. Saunders, 1994.
 3. Mahley RW, Innerarity TL, Rall SC JR, Weisgraber KH. Plasma lipoproteins: apolipoproteins structure and function. *J Lipid Res.* 1984; 25:1277-94.
 4. Ginsberg HN, Le NA, Melish J, *et al.* Effect of a high carbohydrate diet on apoprotein B catabolism in man. *Metabolism.* 1981; 30:347-49.
 5. Davignon J, Gregg RE, Sing CF, Apolipoprotein E. Polymorphism and atherosclerosis. *Atherosclerosis.* 1988; 8:1-21.
 6. Stamler J, Wentworth D, Neaton JD. For the MRFIT Research Group. Is relationship between serum cholesterol and risk of premature death from coronary heart disease continuous and graded? Findings in 356222 primary screenes of the multiple risk Factor Interventional Trial MRFIT. *JAMA.* 1986; 256:2823-8.
 7. Lipid Research Clinics Program. The Lipid Research Clinics Coronary Primary Prevention Trial results II: The Relationship of reduction in incidence of coronary heart disease to cholesterol lowering. *JAMA.* 1984; 251:365-74.
 8. Wong ND, Wilson PWF, Kannel WB. Serum cholesterol as a prognostic factor after myocardial infarction: the Framingham study. *Ann Intern Med.* 1991; 115:687-93.
 9. Lloyd-Jones DM, Larson MG, Beiser A, Levy D. Lifetime risk of developing coronary heart disease. *Lancet.* 1999; 353:89-92.