Original Research Article

Association between Lipid Profile and Lipoprotein(a) levels in individuals with Hypercholesterolemia.

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Abstract

The aim of this study is the association between Lipid Profile and Lipoprotein (a)Lp(a) levels individuals with Hypercholesterolemia (i.e) Total Cholesterol (TC) with >200mg/dLFasting.blood samples were collected from both males and females and were divided into two groups – Group -1 with Total Cholesterol < 200mg/dL (n= 25) and Group - 2 with Total Cholesterol > 200mg/dL (n= 25) . Serum Lp(a) was determined by Particle Enhanced Immunoturbidimetric Assay and Lipid Profile,which includes Total Cholesterol (TC), HDL-Cholesterol (HDL-C) and Triglycerides (TGL) were determined in fully-automated analyser.Non-HDL-Cholesterol and LDL Cholesterol (LDL-C) was calculated by Friedewald's formula. A significant increase in Serum Lp(a) levels , HDL - C ,TGL ,Non- HDL-Cholesterol and LDL-C was observed in Group-2 individuals when compared with Group1.There was significant positive correlation (r) observed between Serum Lp(a) and Total Cholesterol and Serum Lp(a) and LDL-C with r = 0.61 and 0.69 respectively in Group-2 individuals.The subjects with high Cholesterol and high Lp(a) levels are at a high risk of developing Coronary heart disease which causes 40-50% of deaths in India . This study supports the need to intensify lipid management in individuals with high Lp(a) levels.

Keywords: Lipoprotein (a) (Lp(a) , Total Cholesterol (TC) , HDL-Cholesterol (HDL-C) , Triglycerides (TGL) Non-HDL-Cholesterol , LDL Cholesterol (LDL-C) and Coronary heart disease.

Introduction

Lp(a) is a LDL like particle, discovered as a sinking prebeta lipoprotein. This variant lipoprotein fraction contains one molecule of an apolipoprotein B100 and another large protein called apolipoprotein (a)[Apo (a)].Following its discovery Lp(a) was shown in case control studies to be associated with CHD.[1-3]. Elevated Lp(a) levels were proven as a marker of increased cardiovascular risk in numerous epidemiological and genetic studies during the past decades.[4-6].The finding of a curvilinear relationship between systemic levels of LDL cholesterol (LDL-C) and prospective cardiovascular risk in population studies [7] and unequivocal clinical benefit of therapies that lower LDL-C [8] have prompted its central role in the approach to risk prediction and preventive strategies. Hypercholesterolemia particularly with high levels of LDL-C and/or low levels of high-density lipoprotein cholesterol (HDL-C) is an established coronary risk factor that induces endothelial cell dysfunction and impairs collateral vessel growth[9]. Increased Lp(a) confers greater risk for poor coronary collateralization when total cholesterol,LDL-C or non-HDL-C are elevated especially for patients with type 2 diabetes[10].The current study is to observe the association between Lp(a) and Lipids i.e. Total Cholesterol (TC), LDL Cholesterol (LDL-C) and Non-HDL-Cholesterol in individuals with Hypercholesterolemia (Group –2) and in individuals with normal Total Cholesterol (TC) levels (Group -1).

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Materials and methods

This study was conducted in Chalmeda Anand Rao Institute Of Medical Sciences,Karimnagar. The individuals who come first time for the routine health checks with no previous history of high Cholesterol levels and not on any lipid lowering drugs were included in this study. Whether the individuals had Diabetes or Hypertension was not ruled out. The individuals were divided into two groups based on the levels of Total Cholesterol (TC).Group-1 included individuals with Total Cholesterol (TC) < 200 mg/dL(n=25) and Group-2 included individuals with Total Cholesterol (TC) > 200mg/dL (n=25).Venous blood samples were collected from individuals who were fasting over night for minimum of 8 hrs.Serum lipids i.e. Total Cholesterol (TC), HDL-Cholesterol (HDL-C) and Triglycerides (TGL) were analysed in fully automated chemistry analyser. LDL-C was calculated using Friedewald's formula[LDL-C = TC–(HDL-C+TGL÷5)] after considering its limitations. Non HDL-C was calculated by the formula [TC-HDL-C]. Lp(a) measured by Particle Enhanced Immunoturbidimetric Assay in analyser.

Statistical analysis

The statistical analysis was done by SPSS software.Data was expressed as Mean ±SD.Significance of difference between the two groups observed was assayed by using Student 't' test . P-value of < 0.05 was considered to be significant.

Results

Group-1 : The age ranged between 35-69 years with Mean 47±10.3 years and included 10 females and 15 males.

Group-2 : The age ranged between 35-59 years with Mean 45±7.4 years and included 12 females and 13 males.

The statistical data was used to find the association between Serum Lipoprotein(a)Lp(a),Total Cholesterol (TC),HDL-Cholesterol (HDL-

C), LDL Cholesterol (LDL-C) and Non-HDL-Cholesterol in the above mentioned two groups

Table 1 : Demographic details, Lipid profiles and Lp(a) levels in the two groups

Parameters	Group-1 (N=25)		Group-2 (N=25)	
	Range	Mean±SD	Range	Mean±sd
Age(Years)	3 5- 69	47 ± 10.3	35 - 59	45 ± 7.4
Sex	F = 10 M = 15	----	F = 12 M = 13	----
TC (mg/dL)	86 - 195	153 ± 33.8	211 - 490	284 ± 79.2*
TGL (mg/dL)	53 - 308	137 ± 77.5	71 - 365	191 ± 81.3**
HDL-C (mg/dL)	22 - 44	34 ± 7.6	28 - 81	53 ± 14.7*
LDL-C (mg/dL)	45 - 136	91 ± 27	122 - 370	188 ± 70.1*
Non HDL-C (mg/dL)	75 - 160	120 ± 31.3	151 - 341	226 ± 73.3*
Lp(a) (mg/dL)	6 - 29	17 ± 7.2	30 - 122	63 ± 35.5**

NOTE: ** = p- Value <0.005* = p -Value < 0.0001

The table:1 shows that there was significant increase in HDL-Cholesterol , LDL-Cholesterol and Lp (a) levels in the Group-2 with hypercholesterolemia when compared with the Group-1 with normal T.Cholesterol ,with p-Value < 0.0001.

Table 2 : The Coreelation between TC,HDL-C,LDL-C , non-HDL-C and Lp(a) in individuals withT chloesterol

Parameters	TC	HDL-C	LDL-C	Non-HDL C
Lp(a)	r = 0.61 p = 0.0043	r = 0.25 p = 0.28	r = 0.31 p = 0.0008	r = 0.61 p = 0.18

Asignificant positive correlation was observed between T.cholesterol and Lp (a) and between LDL-CholesterolLp(a) levels.

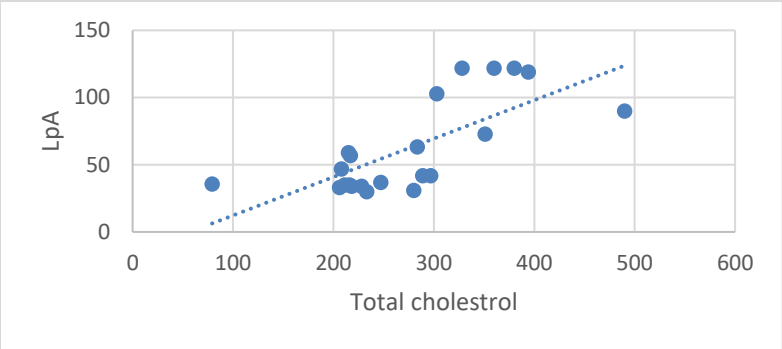

Fig 1: Correlation graph between LpA and Total cholestrol

Correlation graph show a trending line of directly proportional line with significance between LpA and total cholestrol.

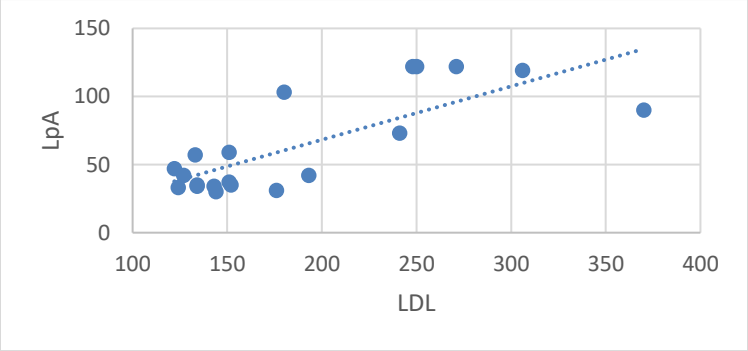

Fig 2: Correlation graph between LpA and LDL

Correlation graph show a trending line of directly proportional line with significance between LpA and LDL.

Discussion

Various risk factors known to cause coronary artery diseases are also known to cause stroke. Among them, dyslipidemia is a well-established risk factor for atherosclerotic coronary artery disease but its relationship to ischemic cerebrovascular disease has remained unclear, perhaps due to heterogeneous nature of stroke etiology[11-13]. Dyslipoprotenemia is well established as being associated with the genesis of ischemic heart disease, but it is not been conclusively demonstrated to be associated with the pathogenesis of atherothrombotic stroke or transient ischemic attack (TIA)[12]. Some of the earlier studies have found TC and TG levels to be elevated in patients with stroke[14]. In the present study there was significant increase in Lp(a) levels in Group-2 when compared to Group-1 .Similarly there was a significant increase in HDL-Cholesterol ,

LDL-Cholesterol and non HDL-Cholesterol in Group-2 when compared with the other Group.There was a significant positive correlation between Total Cholesterol and Lipoprotein(a) with r= 0.61 and p-Value= <0.05 and LDL-Cholesterol and Lipoprotein(a) also showed a significant positive correlation with r= 0.69 and p-Value= < 0.05 in Group-2 . This shows that individuals with high Total Cholesterol and LDL-Cholesterol have a tendency to have hgihLp(a) levels , as shown in the study by Ying Shen,Shuai Chen ,et al Lp(a) was significantly correlated with LDL-C or non-HDL-C, and individuals with high Lp(a) were more likely to have LDL-C > 3.36 mmol/L or non-HDL-C > 4.38 mmol/L, confirming the physiological link between Lp(a) and LDL-C or non-HDL-C. Furthermore, there was a synergistic effect of Lp(a) and LDL-C or non-HDL-C on collateral formation in patients with diabetes. In high tertile of LDL-C or non-HDL-C, diabetic patients with high tertile of Lp(a) had an approximately four fold increased risk of poor coronary collateral-ization compared with those with low tertile of Lp(a)[10].. In the present study even Group-2 showed high Lp(a) levels , because of the major contribution by high LDL-Cholesterol fraction. Lipid profile impairment,especially hypercholesterolemia and high levels of LDL-C and non-HDL, is an established risk factor that induces endothelial cell dysfunction and impairs coronary collateral vessel growth[15]. But in this study no significant correlation was observed between non HDL-Cholesterol and Lp(a) in Group-2 individuals. Alvim et al found that hypercholesterolemia and high non-HDL-C levels were associated with increased arterial stiffness characterized by elevated systolic and pulse blood pressures [16].

Conclusion

The present study demonstrates the assocation between Lp(a) interctions with Cholesterol containing lipids .The observations emphasize the potential importance of Total Cholesterol where main fraction is contributed by LDL-Cholesterol in pointing the individuals at increased risk for Lp(a) mediated disease as well as preventive strategies to mitogate the risk conferred by elevated Lp(a) levels (e.g.Cholesterol lowering).This study substantiates the concept that LDL-reduction with Statin therapy remains the mainstay of pharmacotherapy for dysplipidemia and the percentage reduction in LDL-Cholesterol lowering is strongly correlated with the reduction in atherosclerotic cardiovascular disease risk and events[15,17,18]

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