International Journal of Research in Medical Sciences
Premkumar KS. Int J Res Med Sci. 2017 Feb;5(2):639-645
www.msjonline.org
pISSN 2320-6071 | eISSN 2320-6012
DOI: http://dx.doi.org/10.18203/2320-6012.ijrms20170167

Original Research Article

A study on lipoprotein(a) in health and type-2 diabetes mellitus

Premkumar K. S.*

Department of Biochemistry, Chengalpattu Medical College, Chengalpattu, Tamilnadu, India

Received: 03 November 2016
Revised: 08 December 2016
Accepted: 20 December 2016

***Correspondence:**
Dr. Premkumar K. S.,
E-mail: premkirthu@yahoo.co.in

ABSTRACT

Background: Increased lipoprotein (a) [Lp (a)] concentrations are predictive of coronary artery disease (CAD). Type 2 diabetes mellitus also leads to dyslipidemia, which are known risk factors for CAD. This study was designed to investigate the levels of Lp (a) in type 2 diabetic patients and their association with healthy controls and glycemic control.
Methods: The study included 87 subjects out of which 20 were healthy volunteers. The remaining 67 were patients with type 2 diabetes from which 3 groups were formed 23 formed newly diagnosed group while those on treatment for diabetes were 44 out of which 22 were type 2 diabetics on oral hypoglycemic agents and the other 22 were type 2 diabetics on insulin. Individuals suffering from HT, renal disease, liver disease, thyroid dysfunction, nephrotic syndrome & cardiac disease, alcoholics, smokers or on lipid lowering drugs were excluded. Statistical analysis was done using the pearsons correlation.
Results: Lp(a) levels were found to be significantly increased in the diabetic group irrespective of whether newly diagnosed not on treatment or old cases on treatment with oral hypoglycemic agents or insulin. Lp(a) levels showed no correlation to the degree of glycemic control in these patients. Lp(a) positively correlates with total cholesterol, LDLc and negatively with TGL and VLDLc in diabetics while it does not correlate with any of the lipid parameters in controls
Conclusions: The results of the present study suggest that Lp(a) levels are increased in type 2 diabetic patients. The elevated Lp(a) levels do not reflect the glycemic status and correlates with increase in total cholesterol and LDLc suggesting similar metabolic pathways and the genetic connection for LDL and Lp(a).

Keywords: Cholesterol, LDLc, Lipoprotein(a), Type 2 diabetes, TGL, VLDLc

INTRODUCTION

Lipoprotein (a) is identified as a major risk factor of atherosclerosis in non – diabetic and diabetic patients. It is a well-known fact that diabetic patients have a high risk of cardiovascular disease.[1-3]

In NIDDM patients, as in nondiabetic individuals, atherosclerosis is associated with Lp(a), and Lp(a) is a risk factor for CVD in most studies of NIDDM patients. However, glycemic control has different effects on serum Lp(a).[4,5] In NIDDM, tightened glycemic control does not affect serum Lp(a). Various function like Tissue Repair, Inhibition of fibrinolysis, Effect on Atherogenesis, Lp(a) particles are susceptible to oxidative modification and scavenger receptor uptake, leading to intracellular cholesterol accumulation and foam cell formation which contributes further to atherogenesis.[6,7]

It is said that the atherogenic effect of Lp(a) is due to the cholesterol delivery to the site of injury or to the endothelial cells, blocking of plasmin generation, endothelial cell modulation, smooth muscle cell proliferation and angiogenesis.[8-10] Lp(a) is said to cause

neovascularization atherosclerotic plaque thus contributing to angiogenesis.[11-13]

The pathological effects due to increased Lp(a) was noticed when it exceeded 30 mg/dl.[6,7] Reference plasma concentration of Lp(a) is around 15-20 mg/dl while in asian Indians it is around 30 mg/dl.[14-18] Increased serum Lp(a) is seen in both Type 1 and Type 2 Diabetic Patients.[19] It is determined that Lp(a)is increased in both types of Diabetes Mellitus. The increase was found with Diabetes Mellitus with or without microalbuminuria where increased Lp(a) was found to be an independent risk factor for atherosclerosis.

In certain studies there are no statistical differences between Lp(a) levels of both types of Diabetes mellitus and healthy controls. and it is found to be independent of short term and long term glycometabolic control or the occurrence of microalbuminiuria, neuropatheis or retinopathies. In certain studies Lp(a) levels were not elevated in diabetic patients even in poorly controlled metabolic conditions. It is also found that in Type 1 diabetes mellitus patients, improvement of glycaemic control does not improve plasma Lp(a) concentration regardless of baseline Lp(a) levels and the degree of glycaemic control.

The purpose of the present study was to measure Lp(a) levels in patients with Type II Diabetics and to determine the relationship between Lp(a) and other lipid parameters in Normal and Type 2 Diabetic subjects and to see whether there is any difference in Lp (a) levels between Type 2 Diabetics with good glycemic control and poor glycemic control in our part of the country, this study was done.

Aim and objectives

The work of determining Lp(a) and other associated biochemical parameters namely plasma glucose, Total Cholesterol, Triglycerides, HDLc, VLDLc and LDLc in the blood of healthy and Type 2 diabetics was taken up with the view of establishing the following

- The Blood level of Lp(a) in Health.
- To determine whether Lp(a) levels differs from that in health and in Type 2 Diabetic patients.
- To find out the relationship between Lp(a) with other biochemical parameters in health and the different groups of Type 2 Diabetics patients.

METHODS

The study was carried out on 87 unrelated individuals who have been living in Tamilnadu for 3 generation. Out of the 87 subjects 20 were from apparently healthy volunteers The remaining 67 were diabetic patients Out of 67 diabetic patients selected based on the selection criteria 3 groups of Type 2 diabetics could be arrived at i.e. 23 of them formed the newly diagnosed group while those on treatment were 44 out of which 22 were Type 2 Diabetics on oral hypoglycemic agents and the other 22 were Type 2 diabetics on treatment with insulin. Those individuals who were suffering from diseases like HT, Renal failure, liver failure, thyroid dysfunction, nephrotic syndrome and cardiac pulmonary bypass along with alcoholics and smokers were excluded from the study. None of the subjects were on any drugs with lipid lowering effect.

Blood collection

7ml of blood was drawn from all the above subjects from the anterior cubital vein using sterile disposable syringe. While 1ml blood was collected for blood sugar estimation, 1 ml of clear cell free serum for Lp(a) estimation and rest used for lipid profile testing. Estimation of plasma Glucose was by Glucose Oxidase Peroxidase method, Total Cholesterol, Triglycerides by Enzymatic calorimetric method, HDL by Phosphotungstic acid method, VLDL Cholesterol and LDL Cholesterol were calculated using the Friedewald Equation, Liporotein (a) by Immunoturbidimetric Method Table 1-5.

RESULTS

The levels of Lp(a) and other biochemical parameters namely plasma glucose, Total Cholesterol, TGL, HDLc, LDLc and corrected LDLc in the blood of all the subjects irrespective of the group to which they belong have been compared.

Table 1(a): Comparison of the mean levels of biochemical parameters of newly diagnosed type 2 diabetics with controls.

Control		Pl. Glu mg/dl	S.Lp(a) mg/dl	S.Cholesterol mg/dl	S.TGL mg/dl	S.HDLc mg/dl	S.VLDLc mg/dl	S.LDLc mg/dl	Corrected LDLc mg/dl
Control 20	Mean-SD	77.85± 13.05	18.69± 8.87	181.6 ±24.15	131.1± 55.65	43.35±5.04	26.4±11.21	111.85±25.5	106.23±25.36
New 23	Mean-SD	259.17± 142.48	32.58 25.65	195.52 ±51.38	215.35± 146.74	42.3±9.71	43.35±29.43	110.78±53. 76	100.83±50.14
	P-Value	0.001	0.05	0.840	0.01	0.657	0.01	0.072	0.13
	Significance	HS↑	S↑	NS	HS↑	NS	HS↑	NS	NS

Table 1(b): Comparison of the mean levels of biochemical parameters of type 2 diabetics on OHA with controls.

Control		Pl. Glu mg/dl	S.Lp(a) mg/dl	S.Cholesterol mg/dl	S.TGL mg/dl	S.HDLc mg/dl	S.VLDLc mg/dl	S.LDLc mg/dl	Corrected LDLc mg/dl
Control 20	Mean-SD	77.85± 13.05	18.69± 8.87	181.6± 24.15	131.1± 55.65	43.35± 5.04	26.4± 11.21	111.85± 25.5	106.23± 25.36
OHA 22	Mean-SD	168.23± 130.16	29.68± 18.17	214.36± 30.22	210.91± 100.28	33.41± 7.02	42.14± 20.12	138.36± 36.87	129.46± 33.799
	P-Value	0.001	0.05	0.023	0.001	0.001	0.002	0.05	0.053
	Significance	HS↑	S↑	S↑	HS↑	HS↓	HS↑	S↑	NS

Table 1(c): Comparison of the mean levels of biochemical parameters of type 2 diabetics on insulin with controls.

Control		Pl. Glu mg/dl	S.Lp(a) mg/dl	S.Cholesterol mg/dl	S.TGL mg/dl	S.HDLc mg/dl	S.VLDLc mg/dl	S.LDLc mg/dl	Corrected LDLc mg/dl
Control 20	Mean-SD	77.85± 13.05	18.69± 8.87	181.6± 24.15	131.1± 55.65	43.35± 5.04	26.4± 11.21	111.85± 25.5	106.23± 25.36
Insulin 22	Mean-SD	175.36± 108.62	41.51± 23.38	219.73± 49.52	194.59± 97.167	38.18± 5.989	38.05± 19.06	142.14± 50.52	129.68± 45.926
	P-Value	0.001	0.001	0.024	0.009	0.031	0.01	0.05	0.07
	Significance	HS↑	HS↑	S↑	HS↑	S↓	HS↑	S↑	NS

Table 2(a): Comparison of the mean levels of biochemical parameters of type 2 diabetics on OHA with newly diagnosed type 2 diabetics.

Control		Pl. Glu mg/dl	S.Lp(a) mg/dl	S.Cholesterol mg/dl	S.TGL mg/dl	S.HDLc mg/dl	S.VLDLc mg/dl	S.LDLc mg/dl	Corrected LDLc mg/dl
Control 20	Mean-SD	259.17± 142.48	32.58± 25.65	195.52± 51.38	215.35± 146.74	42.3± 9.71	43.35± 29.43	110.78± 53.76	100.83± 50.14
New 23	Mean-SD	168.23± 130.16	29.68± 18.17	214.36± 30.22	210.91± 100.28	33.41± 7.02	42.14± 20.12	138.36± 36.87	129.46± 33.799
	P-Value	0.047	1.00	0.765	1.00	0.001	1.00	0.222	0.12
	Significance	S↓	NS	NS	NS	HS↓	NS	NS	NS

Table 2(b): Comparison of the mean levels of biochemical parameters of type 2 diabetics on insulin with newly diagnosed type 2 diabetics.

Control		Pl. Glu mg/dl	S.Lp(a) mg/dl	S.Cholesterol mg/dl	S.TGL mg/dl	S.HDLc mg/dl	S.VLDLc mg/dl	S.LDLc mg/dl	Corrected LDLc mg/dl
Control 20	Mean-SD	259.17± 142.48	32.58± 25.65	195.52± 51.38	215.35± 146.74	42.3± 9.71	43.35± 29.43	110.78± 53.76	100.83± 50.14
OHA 22	Mean-SD	175.36± 108.62	41.51± 23.38	219.73± 49.52	194.59± 97.167	38.18± 5.989	38.05± 19.06	142.14± 50.52	129.68± 45.926
	P-Value	0.048	0.869	0.308	1.00	0.357	1.00	0.109	0.114
	Significance	S↓	NS	NS	NS	NS	NS	NS	NS

Table 2(c): Comparison of the mean levels of biochemical parameters of type 2 diabetics on Insulin with type 2 diabetics on OHA.

Control		Pl. Glu mg/dl	S.Lp(a) mg/dl	S.Cholesterol mg/dl	S.TGL mg/dl	S.HDLc mg/dl	S.VLDLc mg/dl	S.LDLc mg/dl	Corrected LDLc mg/dl
Control 20	Mean-SD	168.23± 130.16	29.68± 18.17	214.36± 30.22	210.91± 100.28	33.41± 7.02	42.14± 20.12	138.36± 36.87	129.46± 33.799
Insulin 22	Mean-SD	175.36± 108.62	41.51± 23.38	219.73± 49.52	194.59± 97.167	38.18± 5.989	38.05± 19.06	142.14± 50.52	129.68± 45.926
	P-Value	1.00	0.344	1.00	1.00	0.189	1.00	1.00	1.00
	Significance	NS	NS	NS	NS	NS	NS	NS	NS

Irrespective of the sex the mean obtained in the Control Group has been selected for all the Biochemical Parameters as the Reference Range for the study since there was no appreciable change in the level of any of the

parameters with respect to sex. To find out how far Lp(a) and other parameters varied from the reference range in Type 2 Diabetic groups the Mean and Standard deviation of each parameter in all the 3 groups of Type 2 Diabetics were compared with that of Reference Range in Table 1a-c respectively. To determine the difference in the parameters among the Diabetic groups analysed inter comparison of the results obtained in the different groups of Diabetes was undertaken in Table 2a, 2b and 2c. To get at the overall perspective of the parameters statistical variation between controls and diabetics, the mean and standard deviation of the parameters in the entire group of diabetics obtained have been compared with that of the Reference Range in Table 3.

Table 3: Comparison of the mean levels of biochemical parameters of type 2 diabetics with controls.

		Pl. Glu mg/dl	S.Lp(a) mg/dl	S.Cholesterol mg/dl	S.TGL mg/dl	S.HDLc mg/dl	S.VLDLc mg/dl	S.LDLc mg/dl	Corrected LDLc mg/dl
Control 20	Mean-SD	77.85± 13.048	18.69± 8.87	181.6± 24.154	131.1± 55.645	43.35± 5.04	26.4± 11.208	111.85± 25.504	106.23± 25.356
Diabetics 67	Mean – SD	205.7± 131.605	34.56± 22.88	209.66± 45.404	207.07± 116.024	38.03± 8.485	41.21± 23.204	130.13± 49.081	119.7± 45.404
	P-Value	0.001	0.003	0.01	0.006	0.009	0.007	0.031	0.209
	Significance	HS↑	HS↑	HS↑	HS↑	HS↓	HS↑	S↑	NS

Table 4: Correlation between serum LP(A) and all the other biochemical parameters in controls and diabetics.

		Plasma Glucose		Serum Cholesterol		Serum TGL		Serum HDLc		Serum VLDLc		Serum LDLc		Serum LDL-Corr.	
		Control	Diabetic	Control	Diabetic	Control	Diabetic	Control	Diabetic	Control	Diabetic	Control	Diabetic	Control	Diabetic
Pearsons Correlation	Lp (a)	-0.115	0.036	0.201	0.488**	0.202	-0.294*	-0.012	0.158	0.214	-0.295*	0.099	0.584*	-0.005	0.481**
Sig 2 tailed		0.628	0.773	0.395	0.001	0.393	0.01	0.959	0.201	0.366	0.015	0.677	0.001	0.982	0.001
N		20	67	20	67	20	67	20	67	20	67	20	67	20	67
		NS (p=0.628)	NS (p=0.773)	NS (p=0.395)	+ve HSC (p=0.001)	NS (p=0.393)	-ve HSC (p=0.01)	NS (p=0.954)	NS (p=0.201)	NS (p=0.954)	-ve HSC (p=0.015)	NS (p=0.393)	+ve HSC (p=0.001)	NS (p=0.982)	+ve HSC (p=0.001)

NS=No significant correlation; +ve HSC=Positive highly significant correlation; -ve HSC= Negative Significant Correlation

Table 5: Pearson's correlation coefficient in all subjects (controls + type 2 diabetics).

		Sugar	Cholesterol	TGL	HDLc	VLDLc	LDLc	LDLc- Corr.
Lp(a)	Pearson Correlation	0.161	0.516**	-0.153	0.048	-0.155	0.573**	0.467**
	Sig. (2 – tailed)	0.135	0.001	0.157	0.662	0.150	0.001	0.001
	Significance	NS	HS	NS	NS	NS	HS	HS

Statistical significance has been derived for each parameter in the comparison tables from the p-value obtained which has been calculated using the student t-test.

To obtain the correlation of Lp(a) with lipid parameters Pearson's correlation co-efficient were arrived at for each parameter in control and Diabetics separately and together for the entire 87 subjects analyzed irrespective of the factor whether diabetics or not. The results of Pearson's correlation between Lp(a) and all the other Biochemical Parameters have been tabled for controls and diabetics Table 4 and for all the subjects(Control and Type 2 Diabetics) together in Table 5.

DISCUSSION

The mean level of 18.69±8.87 mg/dl of Lp(a) obtained from controls is well within the Reference Range of less than 30 mg/dl. Similarly even in the kit methodology adopted for its analysis a level of less than 30 mg/dl has been specified as the Reference Range. The different levels of reference range of Lp(a) level is said to be heritable and there is striking difference in its normal levels in various population inspite of the fact that Lp(a) in blood is constant at any stage of life whether it be newborn, adult or oldage.[21] Moreover it has been reviewed that diet can influence Lp(a) levels; hence the varied dietary habits of the different races of people can

also be a contributory factor for the different Reference Range of Lp(a) obtained by each.[22]

Comparison of the mean levels of biochemical parameters of Newly diagnosed Type 2 Diabetics who have not been initiated any treatment for it with that of controls (Table No.1a) shown as S ↑ in Lp(a) (p=0.05) and a HS ↑ of plasma glucose (p = 0.001), TGL (p=0.01) and VLDLc (p=0.01). Similar comparison of Type 2 Diabetics on treatment with Oral hypoglycemic agents with that of controls (Table 1b) reveals in addition to the statistical significance of the former table S ↑ of Total Cholesterol (p=0.023), and LDLc (p=0.05) and HS ↓ of HDLc (p=0.001). Comparison of the mean levels in Type 2 Diabetics on insulin treatment with that of controls (Table 1c) shows that there is HS ↑ of Lp(a) (p=0.001) similar to that of plasma glucose (p=0.001), TGL (p=0.009) and VLDLc (p=0.01) together with a S ↑ of serum Total cholesterol (p=0.024) and LDLc (p=0.05) against a S ↓ of HDLc (p=0.031).

HS increase of plasma glucose in the above Table is natural as the comparison is between diabetics and the non – diabetic healthy. The increase of Lp(a) level in diabetics from its level in healthy controls which is obvious in Table 1a-c is not a surprise for there are several literature evidences that in Diabetics mellitus whether it be in Type 1 or 2 there is increase of Lp(a). The observed increase of Lp(a) in all 3 groups of diabetics analyzed can be attributed to be due to the following reasons :

- As the diabetic groups analyzed belonged to Type 2 Diabetes Mellitus where peripheral resistance to the action of insulin is the main causative factor, hyperinsulinemia will prevail in the above groups. Hence chronic hyperinsulinemia can increase Lp(a) level(23).
- Increase in the rate of synthesis of Lp(a)since Lp(a) level is dependent more on the rate of its synthesis than on its catabolic rate.
- Increased rate of secretion of apoB 100 from the liver will contribute towards the increase of LDL and Lp(a).
- Decreased rate of catabolism of LDL in diabetics(24). As Lp(a) is constituted by apo(a) and LDL, decrease in the catabolism of the latter will be naturally reflected on the level of Lp(a).
- Lp(a) is catabolized by the same receptor by which LDL is catabolized. Hence Lp(a) sharing the same receptor as LDL for its catabolism will be naturally increased LDL has a higher affinity for the receptor than Lp(a). Hence when LDL level is increased in diabetics it will compete with Lp(a) for the receptor.

The most common early alternation of lipoproteins in Type 2 Diabetes is hypertriglyceridemia resulting from evaluation of VLDL concentration, there is a HS ↑ of TGL and VLDLc in all the 3 groups of Type 2 Diabetes Mellitus from their Reference Range. This increase of TGL and VLDLc in Type 2 Diabetes can be due Overproduction of substrates particularly glucose and free fatty acids to liver, Defects in clearance of VLDL triglyceride, Decrease in activity of LPL and Overproduction of VLDL apoB and decrease in its fractional catabolic rate.

The significant increase of LDL cholesterol and Total cholesterol in Type 2 Diabetics can be the result of Defect in LDL clearance which may be due to insulin resistance or deficiency, Reduction in the clearance rate for LDL apoB, Increase in the proportion of small dense triglyceride enriched LDL which has decreased ability to bind to receptors. Nonenzymatic glycation of apoB of LDL which decreases LDL catabolism, Block in cholesterol ester transfer activity from HDL to VLDL and LDL on the other hand decrease in HDL cholesterol in Type 2 Diabetes can be attributed to Increased rate of HDL clearance, Elevated hepatic lipase activity, Decrease in LPL activity and impaired VLDL catabolism.

Absence of any significant change in Total cholesterol, LDLc or HDLc in Newly Diagnosed patients of Type 2 Diabetes which is contrary to the changes observed in patients treated with OHA or insulin can be attributed to the long standing duration of the disease in the latter two groups to that of the former group. The earliest lipoprotein change namely increase of TGL and VLDLc is seen in the newly diagnosed group of Type 2 Diabetic and hence it may be presumed that the subjects belonging to this group have attended the Diabetic OP during the early phase of the disease itself. Therefore even though the plasma glucose level of this group of Type 2 Diabetes Mellitus is higher to the other two groups of treated Type 2 Diabetes Mellitus it has not significantly affected the other lipid parameters. Corrected LDLc does not show any significance in the above 3 comparative tables. The absence of any SS for corrected LDLc inspite of a S ↑ of Lp(a) in Table 1a, 1c suggests that the LDLc increase is the result of the additional cholesterol of Lp(a). So when this fraction is deducted from LDLc to give the corrected LDL the SS disappears.

In comparison of the 3 groups of Diabetics with each other in Table 2a-c reveals that plasma glucose levels are lower in both the treated groups to that in the newly diagnosed groups, the degree of lowering being statistically significant. Similarly the levels of HDLc are lower in Type 2 diabetes treated with OHA than to its level in the newly diagnosed group. However statistical significance to the extent of HS has been obtained only between the HDLc of the Diabetic group on OHA and the newly diagnosed group. The glycaemic control achieved by the diabetic patients of this study is not sufficient to increase the HDLc level to the level in the Newly Diagnosed group which is near normal.

The mean level of the assessed parameters of all the 67 diabetic patients compared with that of controls (Table 3) shows a statistical increase in the level of all the

parameters in diabetics except LDL which is just significant.

On analysis of the correlation of Lp(a) with other parameters we find that Lp(a) does not correlate to the plasma glucose and that the degree of glycaemic controls does not influence plasma Lp(a) levels either in controls or any of the Diabetic groups analyzed. This finding correlates well to the study of A Perez et al, though his work has been carried out in Type 1 diabetic patients.[25] The absence of any significant correlation between Lp(a) and Blood Sugar in Control and Diabetics is evident in Table 4.

Correlation of Lp(a) with other lipid parameters has revealed a positive HS (p=0.001) correlation between Lp(a) and Total cholesterol in Diabetics (Table 4). This was however absent in controls (Table 4) of the study. On the other hand TGL (p=0.01) showed a negative correlation with Lp(a) in Diabetics (Table 4) with absence of any correlation between the two in controls (Table 4). Among the fractions of cholesterol in the various lipoproteins HDLc does not correlate to Lp(a) either in controls or Diabetics (Table 4); but VLDLc (p=0.015) and LDLc (p=0.001) have negative S correlation and positive HS correlation respectively with Lp(a) in the Diabetics (Table 4) which is absent in their counterpart controls (Table 4). Corrected LDL (p=0.001) also has a similar correlation as that of LDLc with Lp(a) (Table 4). However the correlation of levels of Lp(a) in the entire 87 subjects irrespective of the fact Diabetic or not proves that only Total cholesterol (p=0.001), LDLc (p=0.001) and corrected LDL (p=0.001) have a positive HS correlation with Lp(a) (Table 5). As LDLc is the main lipid fraction of LDL and as LDL is a component of Lp(a) the author is not surprised to see the positive HS correlation of LDLc or corrected LDLc. Moreover as LDLc is the major fraction of the Total cholesterol the HS positive correlation of Lp(a) with LDLc is also reflected on Total cholesterol.

Since the level of the main biochemical parameters of this study namely Lp(a) differs statistically by a HS degree between controls and diabetics attempt is made to arrive at a cut off level for this parameter between the two groups. For this purpose the levels of Lp(a) in the 20 controls and 18 of each group of Type 2 diabetes are plotted. Various cut off levels have been selected between controls and the diabetic and the sensitivity, specificity, positive predictive value and negative predictive value have been calculated. From this it is clear that 25 mg/dl of Lp(a) is the most appropriate cut off level to demarcate between controls and Type 2 Diabetes.

CONCLUSION

From the results on the study on 87 subjects consisting of 20 control, 23 Type 2 diabetic patients who were newly diagnosed and not on any treatment, 22 Type 2 diabetic patients on oral hypoglycemic agents and 22 type 2 diabetic patients on oral hypoglycemic agents and 22 type 2 diabetic patients on insulin therapy in whom serum Lp(a) along with plasma glucose, serum total cholesterol, serum triglycerides, serum HDLc were analyzed and VLDLc, LDLc and corrected LDLc were calculated the following facts are revalued.

- Reference range for serum Lp(a) in the study is 18.69±8.87. Difference in sex does not alter the RR of Lp(a).
- Serum Lp(a) is elevated in Type 2 diabetics. The increase is found in Diabetic irrespective of whether newly diagnosed not on treatment or old cases on treatment with OHA or insulin.
- Serum Lp(a) does not correlate to plasma glucose i.e. to the level of glycaemic control.
- Serum Lp(a) does not correlate with any of the lipid parameters in controls.
- Serum Lp(a) positively correlates with Total Cholesterol, LDLc, and correlated LDLc and negatively with TGL and VLDLc in diabetics.
- The most appropriate cut off level of Lp(a) between controls and diabetics is 25 mg/dl.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: The study was approved by the Institutional Ethics Committee

REFERENCES

1. Utermann G, Weber W. Protein Composition of LP (a) lipoprotein from human plasma. FEBS let. 1983;154:357-61.
2. Berg K. Lp (a) Lipoprotein: an overview. Chem Phys Lipids. 1994:67- 68:9-16.
3. Ralph L. Nachman MD. Lipoprotein (a): molecular mischance in the microvasculature - IRCULATION. 1997;96:2485-7.
4. de Rijke YB, Jürgens G, Hessels EM, Hermann A, van Berkel TJ. In vivo fate and scavenger receptor recognition of oxidized lipoprotein[a] isoforms in rats. J Lipid Res. 1992;33(9):1315-25.
5. Chapman MJ, Huby T, Nigon F, Thillet J. Lipoprotein (a): implication in atherothrombosis. Atherosclerosis. 1994;110 (Suppl):S69-75.
6. Hiraga T, Kobayashi T, Okubo M, Nakanishi K, Sugimoto T, Ohashi Y, et al. Prospective study of lipoprotein(a) as a risk factor for atherosclerotic cardiovascular disease in patients with diabetes. Diabetes Care. 1995;18(2):241-4.
7. Leus FR, Leerink CB, Prins J, van Rijn HJ. Influence of Apo(a) phenotype on Lp(a) qualification- Evaluation of 3 methods- Clinical Biochemistry. 1994;(27):449-55.
8. Paul M, Ridker MD. A prospective study of Lp(a) and the risk of Myocardial Infarction. JAMA. 1993;2710(18):n2195-99.

9. Karmansky I, Gruener N. structure and Possible biological roles of Lp (a). Clinical Biochemistry. 1994;27(3):157-62.
10. Hiraga T, Shimada M, Okubo M, Nakanishi K, Kobayashi T, Murase T. Lipoprotein (a) is an independent risk factor for multiple cerebral infarctions. Atherosclerosis. 1996;122:29-32.
11. Enas EA, Dhawan J, Petkar S. Coronary artery disease in Asian Indians: lessons learnt and the role of lipoprotein(a). Indian Heart J. 1997;49(1):25-34.
12. Wright LC, Sullivan DR. Muller M, Dyne M, Tattersall MHN, Mountford CE. Elevated apolipoprotein (a) levels in cancer patients. Int J Cancer. 1989:43:241-4.
13. Engler H, Riesen W. Effect of Thyroid function on concentration of lipoprotein(a) . Clin. Chem. 1993;39:246609.
14. Feely J, Barry M, Keeling PW, Weir DG, Cooke T. Lipoprotein(a) in cirrhosis. BMJ. 1992;304:545-6.
15. Kung AW, Pang RW, Lauder I, Lam KS, Janus ED. Changes in serum Lipoprotein(a) and Lipids during treatment of hypothyroidism. Clin. Chem. 1995:41:226-31.
16. Relimpio F. High Lp(a) levels in type 1 and 2 Diabetic patients with microalbuminuria. Diabetes Care. 2000;20(12):1921-2.
17. Kaysen GA. Plasma Composition in Nephrotic Syndrome. Am J Nephrol. 1993:13:347-54.
18. Kamanna VS, Roh DD, Kirschenbaum MA. Editorial. Atherogenic lipoproteins-Mediators of Glomerular injury. Am J Nephrology. 1993;13:1-5.
19. Mohan V. Lp(a) is an independent risk factor for coronary artery disease in NIDDM patients in South India.Diabetes Care. 1990;21(11):1819-23.
20. Angela M, Scanu MD. Atherothrombogenecity of Lipoprotein (a) : The Debate - Am.J.Cardiology. 1998;82:262-32.
21. Elliot S, Barnathan MD. Has little Lp(a) shrunk? JAMA. 1993;270(18):2224-5.
22. Nilausen K, Meinertz H. Lipoproteinm (a) and Dietary Proteins: Casein lowers Lipoprotein(a) concentrations as compared with soy protein. Amer J Clin Nut. 1999;69:3:419-25.
23. Heller FR, Jamart J, Honore P, Dermeet G. Serum Lipoprotein (a) in patients with Diabetes Mellitus. Diabetes Care. 1998;16(5):819-23.
24. Howard BV, Abbott WGH, Bettz WF. Integrated study of low density lipoprotein metabolism and very low density lipoprotein metabolism in non-insulin dependent diabetes. Metabolism 1987,36:870-7.
25. Pérez A, Carreras G, Caixàs A, Castellví A, Caballero A, Bonet Ret al. Plasma lipoprotein(a) levels are not influenced by glycemic control in Type I diabetes. Diabetes Care. 1998;21(9):1517-20.

Cite this article as: Premkumar KS. A study on lipoprotein(a) in health and type-2 diabetes mellitus. Int J Res Med Sci 2017;5:639-45.