



## **Comparative Analysis of Serum Lipoprotein (a) in Hypothyroid and Healthy Subjects**

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### **Authors' contributions**

*This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.*

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### **ABSTRACT**

This study is taken up to estimate and compare the level of serum Lp(a) in hypothyroid patients and in healthy controls. A total of 50 hypothyroid patients within aged group 20-60 years and total of 50 healthy controls within 20-60 years were enrolled in the study after taking written consent. Thyroid profile and Lp(a) were measured by CLIA and immune turbidimetric method respectively. Data collected was analysed using Stata version 14.1 software. Result shows an increased level of Lp(a) among hypothyroid patients when compared to healthy controls.

*Keywords: Hypothyroid; lipoprotein (a); healthy controls; cardiovascular diseases.*

### **1. INTRODUCTION**

Lipoprotein (a) is LDL like particle formed by the association of highly polymorphic glycosylated apolipoprotein (a) through a disulphide bond with

apolipoprotein B100, [1] the classic protein moiety of LDL [2]. It was discovered by Kare Berg in 1963 [3]. Genetic variation of the Lp(a) genes is the main determinant of Lp(a) serum levels but non genetic factors could also affect its

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concentration. Several studies have shown the influence of diet, drugs and hormones on Lp(a) levels. Some authors consider Lp (a) as an independent risk factor for coronary and brain artery atherosclerosis in Caucasian, Chinese, African and Indian population. Lp(a) concentrations (reported as  $\geq 300$  mg/L or  $\geq 30$  mg/dL) and the risk of suffering coronary events, peripheral artery disease, cerebrovascular disease, and the early development of atherosclerosis in children and adolescents [4,5]. In a nutshell 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 [6].

Hypothyroidism is associated with various metabolic abnormalities, due to the effects of thyroid hormones on nearly all major metabolic pathways. It causes derangement of wide range of parameters in lipid profile, hemodynamic changes, endothelial dysfunction, coagulation disturbances, hormonal and metabolic changes, leading to various quantitative and or qualitative changes of triglycerides, phospholipids, cholesterol, and lipoproteins including LDL-Cholesterol, HDL-Cholesterol, lipoprotein (a), apolipoprotein A1, and apolipoprotein B. The

present study was designed to determine the Lp(a) levels among the hypothyroid patient and healthy control and to compare the same.

## 2. MATERIALS AND METHODS

The sample size of this study includes 100 subjects involving 50 hypothyroid patients and 50 healthy control reporting to Medicine OPD, Sree Balaji Medical College and Hospital, Chromepet, were enrolled in the study.

After obtaining informed consent from patient, a detail history was taken followed by laboratory investigation as under:

- Estimation of Lp(a) by immuno-turbidimetry
- Estimation of serum FT3, FT4, TSH by Chemiluminescent immunoassay.

The data was entered in software using Stata 14.1 version, where mean, standard deviation, correlation coefficient and percentage were calculated and results were obtained.

## 3. RESULTS

The Lp (a) levels in different age categories in test samples were shown in Fig. 1.

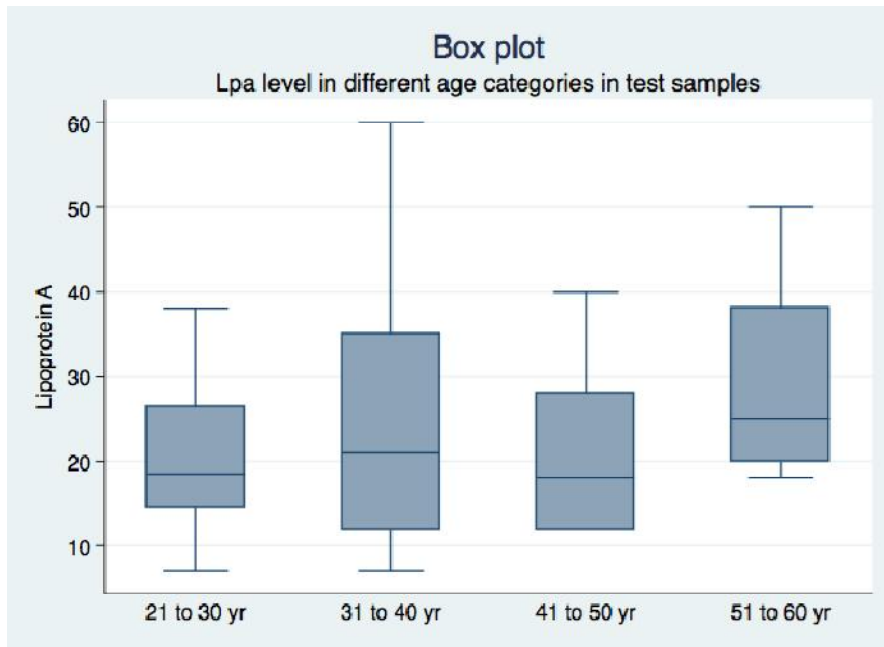
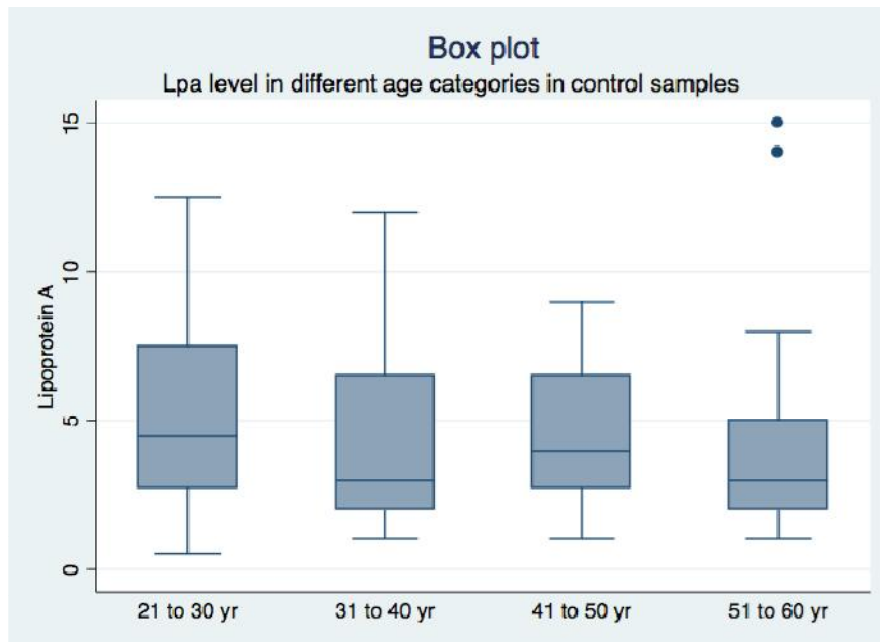


Fig. 1. Box plot showing median, quartiles, and whiskers of Lp (a) levels in different age groups in test samples

**Table 1. Descriptive statistics of case and control**

Group	Mean	Std. Err.	Std. Dev.	[95% Conf. Interval]		t-value
<b>FT3</b>						
Test	2.420	0.075	0.527	2.272	2.568	-7.44**
Control	3.479	0.121	0.858	3.238	3.720	
<b>FT4</b>						
Test	0.841	0.026	0.187	0.788	0.893	-8.38**
Control	1.639	0.092	0.647	1.458	1.821	
<b>TSH</b>						
Test	35.715	3.558	25.156	28.656	42.775	9.29**
Control	2.633	0.142	1.001	2.352	2.914	
<b>Lp(a)</b>						
Test	23.120	1.714	12.118	19.720	26.520	10.24**
Control	4.780	0.522	3.691	3.744	5.816	



**Fig. 2. Box plot showing median, quartiles, and whiskers of Lp (a) levels in different age groups in control samples**

**4. DISCUSSION**

This study was done on known cases of hypothyroidism, and healthy individuals taken as controls. Between the study group, serum Lp(a) were significantly increased in hypothyroid patients when compared to controls. Serum Lp(a) levels among hypothyroid patients (23.120±12.118) were significantly higher than control subjects (4.780±3.691) with t-value = 10.2375 (p < 0.001). It was also observed that serum Lp(a) levels were significantly increased in all age groups and in both genders of hypothyroid patients when compared to control,

which suggested the influence of thyroid hormones on Lp(a) metabolism. The potential association between Lp(a) and thyroid function status in the general population might be regarded as an important aspect for cardiovascular risk prediction and prevention. This hypothesis was supported by De Bruin et al. [7] who demonstrated an almost perfect correlation between free thyroxine index and Lp(a).

Cristina Hernandez et al. have stated that serum Lp(a), level is dependent more on the rate of synthesis than on its catabolic rate [8]. In

hypothyroid state, there is an increase of LDL-C and apo B levels, which may be used in the Lp(a) synthesis, [9] and also there is decreased rate of catabolism of LDL. Since Lp(a) is constituted by apo(a) and LDL, decrease in the catabolism of LDL will be naturally reflected on the level of Lp(a). Moreover, it has been reviewed that 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 because it has been found by Cristina Hernandez et al. [8] that LDL has a higher affinity for the receptor than Lp(a). Hence when LDL level is increased in Hypothyroidism it all the more will compete with Lp(a) for the receptor.

## 5. CONCLUSION

The results of this study provide ample evidence that the levels of Serum Lp(a) are increased in hypothyroid patients when compared to healthy controls. As an outcome of this study, a recommended screening can be advised to hypothyroid patients to estimate Lp(a) level which may assist in early treatment and prevent them from developing cardiovascular diseases.

## CONSENT AND ETHICAL APPROVAL

As per university standard guideline patients' informed written consent and ethical approval has been collected and preserved by the authors.

## COMPETING INTERESTS

Authors have declared that no competing interests exist.

## REFERENCES

1. Utermann G, Weber W. Protein composition of Lp (a) lipoprotein from human plasma. FEBS Lett. 1983;154(2): 357–61.
2. Berg K. Lp (a) lipoprotein: An overview. Chem Phys Lipids. 1994;67:9–16.
3. Berg K. Lp(a) lipoprotein: An overview. In: Scanu AMBT-L (A), editor. Lipoprotein (A) [Internet]. Elsevier. 1990;1–23. Available:<http://www.sciencedirect.com/science/article/pii/B9780126209907500048>
4. Souki-Rincón A, Urdaneta J, Mengual E, Torres D, Cano-Penalosa R, Garcia-Camacho D, et al. Increased levels of lipoprotein (a) are related to family risk factors of cardiovascular disease in children and adolescents from Maracaibo, Venezuela. In: American Journal of Therapeutics. LWW. 2008;403–8.
5. Bermúdez V, Torres Y, Mejías J, Nava A, Añez R, Toledo A, et al. Niveles séricos de Lp (a) y su comportamiento en el estado Zulia: 10 años de investigación. Rev Latinoam Hipertens. 2011;6(4).
6. Nachman RL. Molecular mischief in the microvasculature. Circulation. 1997;96: 2485–7.
7. De Bruin TW, Van Barlingen H, van Linde-Sibenius Trip M, Van Vuurst De Vries AR, Akveld MJ, Erkelens DW. Lipoprotein (a) and apolipoprotein B plasma concentrations in hypothyroid, euthyroid, and hyperthyroid subjects. J Clin Endocrinol Metab. 1993;76(1):121–6.
8. Hernández C, Chacón P, García-Pascual L, Simó R. Differential influence of LDL cholesterol and triglycerides on lipoprotein (a) concentrations in diabetic patients. Diabetes Care. 2001;24(2):350–5.
9. Gaubatz JW, Nava MN, Guyton JR, Hoffman AS, Opekun AR, Hachey DL, et al. Metabolism of apo (a) and apoB-100 in human lipoprotein (a). In: Drugs affecting lipid metabolism. Springer. 1993;161–7.

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