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# Correlation of serum lipoprotein with various defining parameters of metabolic syndrome in obese adolescents

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## Abstract

**Introduction:** High Lp(a) in youth also increases the risk of future atherosclerotic CVD, ischemic stroke, and possibly venous thromboembolic events and MetS has been associated with increased levels of apolipoprotein B (apoB) and low-density lipoprotein oxidation (OxLDL)

**Methodology:** 150 subjects were examined for anthropometric parameters and biochemical investigations as per the guidelines of IDF. For BMI, of 19 year old adolescents, WHO classification of obesity was used. Estimation of serum lipoprotein (a) was done by Latex turbidimetry method.

**Results:** Mean serum Lp(a) was significantly higher in obese adolescents i.e. 11.67±4.87 as compared to mean serum Lp(a) in healthy adolescents' i.e 4.47±3.33

**Conclusion:** The present study concludes that the obese adolescents are at a higher risk of developing cardiovascular abnormalities because of increased level of Lp(a).

**Keywords:** CVD, apoB, OxLDL.

## Introduction

Obesity is the most important risk factor for cardiovascular disease (CVD) and is often clustered with additional metabolic abnormalities including hypertension, dyslipidemia, and insulin resistance.(1) These CVD risk factors tend to cluster, not only in adults, but more recently in children. Childhood obesity is also an early risk factor for adult morbidity and mortality and 85% of obese children become obese adults. The common cluster of major determinants of CVD led to the metabolic syndrome (MetS). The IDF suggests that only children above 10 years of age should be examined for MetS, whereas in younger individuals, WC measurement alone should be used for screening.(2) The current paradigm of MetS was established by Reaven and colleagues in 1988, originally termed

Syndrome X.(3) The presence of central obesity, hyperglycemia, hypertension (HTN), and dyslipidemia (DLP) are the key risk factors for MetS.(4)(5) MetS has been associated with increased levels of apolipoprotein B (apoB) and low-density lipoprotein oxidation (OxLDL) and with an increased risk of cardiovascular disease and non-alcoholic fatty liver disease.(6)

Lp(a) is a low-density Lipoprotein (LDL)-like particle in which apo B is covalently bound by a single disulfide bond to a glycoprotein apolipoprotein(a) (apo(a). High Lp(a) in youth also increases the risk of future atherosclerotic CVD, ischemic stroke, and possibly venous thromboembolic events. All these efects are markedly increased in the presence of high low-density lipoprotein cholesterol (LDL-C) or low high-density lipoprotein cholesterol (HDL-C)(7)

Studies in rabbits have also demonstrated accumulation of injected human Lp(a) in balloon-injured and atherosclerotic arteries.The retention of Lp(a) in the arterial wall is likely due to apo(a)'s affinity for extracellular matrix proteins. Once deposited, both intact Lp(a) and apo(a) fragments can elicit a range of biological activities that fuel the development of atherosclerosis(8)

Hence early detection of Lp(a) biomarker is essential in obese adolescent to prevent and treat obesity and its associated metabolic abnormalities. Diagnosis of which provides the pediatrician with the most evidencebased methods for addressing cardiometabolic risk factor clustering (MetS) in adolescence. In the light of above stated the present study aims at finding the association of Lp(a) protein with MetS disease in obese adolescents.

**Methodology**

The present study was conducted in the Department of Biochemistry, Dr S. N. Medical College and its associated group of hospitals, Jodhpur (Rajasthan). The subjects selected for the study were grouped as follows:

- Group 1- Healthy adolescents (n=75)
- Group 2- Obese adolescents (n=75)

Healthy and obese adolescents aged between 10-19 years of either sex were included in the study. Patients with history of infection and chronic disease, type II diabetes mellitus, familial hyperlipidemia, hypertension, genetic disorders, growth hormone deficiency, hypothyroidism were excluded from the study. An informed consent was taken from all the subjects or their parents or their guardian who participated in the study for physical examination and biochemical procedures after apprising them the nature and objective of the study.

**Physical examination and Anthropometry**

Each subject was examined for anthropometric parameters and biochemical investigations as per the guidelines of IDF (2007). In that report, the IDF recommended that pediatric metabolic syndrome be based on the adult IDF definition but that it should only apply to children 10 years and older and that, among those between10 and 16 years of age, the 90$^{th}$ percentile for waist circumference or adult cut point (which ever was lower) should define abdominal obesity. The IDF stated that for those 16 years and older, adult criteria should apply.

Height was measured in meters (without footwear) by using a standard measuring tape. Weight was measured on electronic weighing machine to the nearest 50 grams with children bare foot and wearing light clothing. Waist circumference in Centimeters, measured at a point midway between lower margin of the rib cage and the highest point of the iliac crest, in the standing position with the abdomen relaxed, arm hanging by the side and the feet together using a standard measuring tape. Hip

circumference in centimetres, at the level of greater trochentar in standing position with the arm hanging by the side using a standard measuring tape. Blood Pressure was recorded in the right arm of the relaxed, seated subject**.**

The body mass index was calculated from the height and weight.

$$\text{BMI}\left(\frac{\text{kg}}{\text{m}^2}\right) = \frac{\text{Weight(kg)}}{\text{Height(m2)}}$$

For the BMI, using IAP (Indian Pediatrics) growth charts for 5-18 year old Indian children, approach 3rd, 5th, 10th , 25th ,50th ,23 adult equivalent (as overweight cut off), and 27 adult equivalent (as obesity cut off) percentiles. For BMI, of 19 year old adolescents, WHO classification of obesity was used,

Estimation of serum glucose was done by enzymatic glucose oxidase- peroxidase endpoint method. Estimation of serum cholesterol was made by enzymatic chod-pap endpoint method. Estimation of serum triglyceride was done by enzymatic gpo/pap endpoint m method. Estimation of serum high density lipoprotein-c (HDL-c) and low density lipoprotein-C (LDL-C) was done by direct homogenous method. Estimation of Serum VLDL was done by Friedwald's formula

Estimation of serum lipoprotein (a) was done by Latex turbidimetry method.(9)

Table 1: Basic characteristics in group 1 and 2

| Parameters | Group-1 (Healthy Adolescents) Mean±SD | Group-2 (Obese Adolescents) Mean±SD | Group-1 vs Group-2 p value |
|---|---|---|---|
| Height | 1.54±0.09 | 1.62±0.09 | <0.0001(HS) |
| Weight | 52.06±10.67 | 76.78±11.02 | <0.0001(HS) |
| BMI | 21.61±1.90 | 32.64±4.30 | <0.0001(HS) |
| Waist Circumference | 67.32±4.52 | 101.81±11.80 | <0.0001(HS) |
| Hip Circumference | 85.97±6.47 | 97.28±8.27 | <0.0001(HS) |
| Waist to Hip Ratio | 0.79±0.04 | 1.05±0.10 | <0.0001(HS) |
| Systolic Blood Pressure | 119.48±1.78 | 133.94±5.57 | <0.0001(HS) |
| Diastolic Blood Pressure | 78.72±2.43 | 84.65±3.03 | <0.0001(HS) |

Table 2: Biochemical parameters in group 1 and 2

| Parameters | Group-1 (Healthy Adolescents) Mean±SD | Group-2 (Obese Adolescents) Mean±SD | Group-1 vs Group-2 p value |
|---|---|---|---|
| Fasting Blood Glucose | 81.81±8.17 | 107.08±13.21 | <0.0001(HS) |
| Serum Total Cholesterol | 116.57±13.17 | 189.18±32.12 | <0.0001(HS) |
| Serum Triglycerides | 80.12±13.09 | 134.06±25.38 | <0.0001(HS) |
| Serum HDL | 46.96±5.80 | 35.28±9.07 | <0.0001(HS) |
| Serum LDL | 90.66±9.16 | 109.77±18.38 | <0.0001(HS) |
| Serum VLDL | 16.05±2.60 | 26.77±5.11 | <0.0001(HS) |
| Serum Lp(a) | 4.47±3.33 | 11.67±4.87 | <0.0001(HS) |

Graph 1: Mean serum lp(a) (mg/dl) of subjects studied

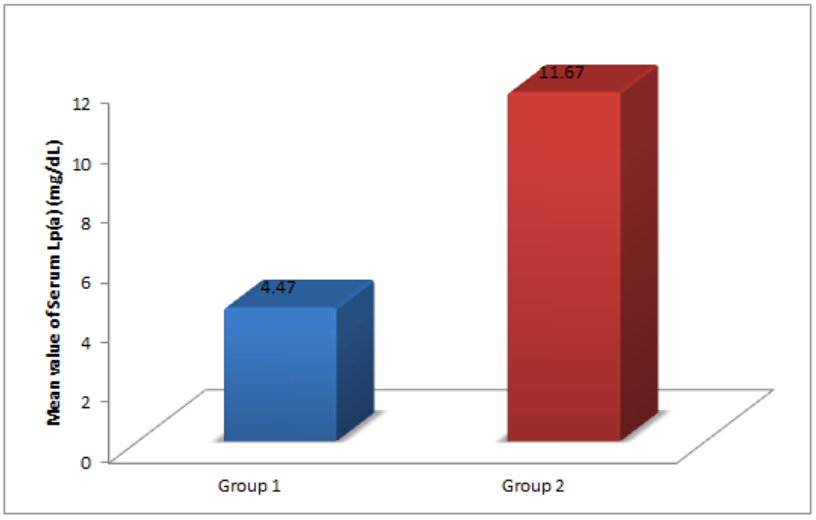


Table 3: Correlation of serum Lp(a) with various defining parameters of metabolic syndrome in group 1-healthy adolescents (10-19 years)

| Defining criteria | Serum Lp (a) | |
|---|---|---|
| | r value | p value |
| BMI | 0.198 | 0.088 |
| Waist circumference | 0.179 | 0.122 |
| Waist-Hip Ratio | 0.144 | 0.217 |
| Systolic blood pressure | 0.021 | 0.857 |
| Diastolic blood pressure | 0.032 | 0.781 |
| Serum Triglyceride | 0.107 | 0.359 |
| Serum HDL | -0.019 | 0.866 |
| Fasting Blood Glucose | 0.158 | 0.175 |

Table 4: Correlation of serum Lp(a) with various defining parameters of metabolic syndrome in group 2-obese adolescents (10-19 years)

| Defining criteria | Lp(a) | |
|---|---|---|
| | r value | p value |
| BMI | 0.265 | 0.021* |
| Waist circumference | 0.244 | 0.034* |
| Waist-Hip Ratio | 0.237 | 0.040* |
| Systolic blood pressure | 0.069 | 0.554 |
| Diastolic blood pressure | 0.126 | 0.281 |
| Serum Triglyceride | 0.291 | 0.011* |
| Serum HDL | -0.039 | 0.738 |
| Fasting Blood Glucose | 0.006 | 0.952 |

Pearson's correlation test applied, * $p < .05$ (Significant)

**Discussion**

MetS is an increasingly international common cause of morbidity and mortality and has been linked with many risk factors as well as numerous postulated pathophysiologic mechanisms. The most commonly described mechanisms result in insulin resistance, together with a low-grade proinflammatory, prothrombotic, and oxidative physiologic state(10) Childhood obesity is associated with a higher chance of adult obesity, consequent disability and early mortality. Novel biochemical markers such as lipoprotein (a) (Lp(a)), uric acid, fibrinogen and homocysteineare increasingly being used to determine CVD related morbidity and mortality.(11)

In present study we observed that serum Lp(a) level is high in obese children and adolescents than non obese children and adolescents which also showed significant relation with BMI, waist circumference, waist to hip ratio and serum triglycerides .Marie-Helene Gannage-Yared et al (7) reported high level of Lp(a) and its significant positive correlation with BMI and also found Lp(a) relationship with lipid parameters; it was positive for non-HDL-C and absent for triglycerides and HDL-C. and suggested that Lp(a) should be measured in children and adolescents with high cardiovascular risk. Similarly to our study, the 3rd NHANES study reported higher numbers of youth with elevated Lp(a) in the subgoups with a high BMI. In another study, Glowinska et al (12)found that young obese, hypertensive and diabetic patients have a Lp(a) twice as higher than in control group.

Conversely T Chandrashekar et al(11) also reported there was no significant difference of serum Lp(a) levels in obese children and control group. Debora Larissa Rufino Alves et al (13)assessed BMI, Lp(a) and ultra sensitive CRP in children and adolescents and reported a weak correlation between BMI and Lp(a) , suggesting that this may have occurred due to the amplitude of results or due to the wide range of age. Sushma Sharma et al (14) carried out a cross sectional analysis on overweight and obese children and reported that Lp(a) shows strong positive relation with HDL and this association is not influenced by other lipoprotein subclasses or by the degree of obesity and concluded

that Lp(a) is not an independent risk factor for CVD in African American children. In a Taiwanese study none of the anthropometric measures (body weight, waist and hip circumference) were significantly correlated with Lp(a) levels .(15)

Treatment of MetS involves both behavioral and pharmacotherapeutic interventions aimed at reducing obesity, glucose abnormalities, hypertension, and dyslipidemia. Obesity treatment is grounded in lifestyle modification, and early treatment of obesity in childhood and adolescence is recommended as the first-line approach to reducing cardiometabolic risk(16)

**Conclusion**

In the present study the Lp(a) in obese adolescents was found to be significantly higher as compared to healthy adolescents. This concludes that the obese adolescents are at a higher risk of developing cardiovascular abnormalities. Therefore, these children must be targeted for life style and dietary modification, and if necessary therapeutic interventions must be initiated to arrest further progression of these risk factors.

**References**


1. Körner A, Kratzsch J, Gausche R, Schaab M, Erbs S, Kiess W. New predictors of the metabolic syndrome in children - Role of adipocytokines. Pediatr Res. 2007;61(6):640–5.
2. Reisinger C, Nkeh-Chungag BN, Fredriksen PM, Goswami N. The prevalence of pediatric metabolic syndrome—A critical look on the discrepancies between definitions and its clinical importance. International Journal of Obesity. 2021 Jan;45(1):12-24.
3. Higgins V, Adeli K. Pediatric metabolic syndrome: pathophysiology and laboratory assessment. Ejifcc. 2017 Feb;28(1):25.
4. Al-Qawasmeh RH, Tayyem RF. Dietary and lifestyle risk factors and metabolic syndrome: Literature review. Curr Res Nutr Food Sci. 2018;6(3):594–608.
5. Tandona N, Garga MK, Singh Y, Marwaha RK. Prevalence of metabolic syndrome among urban Indian adolescents and its relation with insulin resistance (HOMA-IR). J Pediatr Endocrinol Metab. 2013;26(11–12):1123–30.
6. Cornier M, Dabelea D, Hernandez TL, Lindstrom RC, Steig AJ, Stob NR, et al. The Metabolic Syndrome. 2008;29(7):777–822.
7. Gannagé-Yared MH, Lahoud C, Younes N, Chedid R, Sleilaty G. Prevalence and status of Lipoprotein (a) among Lebanese school children. Sci Rep [Internet]. 2020;10(1):1–8. Available from: https://doi.org/10.1038/s41598-020-77689-5
8. McCormick SP. Lipoprotein (a): biology and clinical importance. The Clinical Biochemist Reviews. 2004 Feb;25(1):69.
9. Macy EM, Hayes TE, Tracy RP. Variability in the measurement of C-reactive protein in healthy subjects: implications for reference intervals and epidemiological applications. Clinical chemistry. 1997 Jan 1;43(1):52-8.
10. McCracken E, Monaghan M, Sreenivasan S. Pathophysiology of the metabolic syndrome. Clin Dermatol [Internet]. 2018;36(1):14–20.
11. Chandrasekhar T, Suchitra MM, Pallavi M, Srinivasa Rao PVLN, Sachan A. Risk factors for cardiovascular disease in obese children. Indian Pediatr. 2017;54(9):752–5.
12. Głowińska B, Urban M. Selected cytokines (Il-6, Il-8, Il-10, MCP-1, TNF-alpha) in children and adolescents with atherosclerosis risk factors: obesity, hypertension, diabetes. Wiadomosci Lekarskie

(Warsaw, Poland: 1960). 2003 Jan 1;56(3-4):109-16.

13. Alves DL, de Farias CR, da Costa IF, da Silva Simões MO, Medeiros CC, de Carvalho DF. Lipoprotein (a) and Ultrasensitive C-Reactive Protein in Overweight Adolescents. Health. 2014 Sep 30;6(17):2349.
14. Sharma S, Merchant J, Fleming SE. Lp (a)-cholesterol is associated with HDL-cholesterol in overweight and obese African American children and is not an independent risk factor for CVD. Cardiovascular diabetology. 2012 Dec;11(1):1-7.
15. Chiang JK, Lai NS, Chang JK, Koo M. Predicting insulin resistance using the triglyceride-to-high-density lipoprotein cholesterol ratio in Taiwanese adults. Cardiovascular diabetology. 2011 Dec;10(1):1-6.
16. Magge SN, Goodman E, Armstrong SC, Daniels S, Corkins M, De Ferranti S, et al. The metabolic syndrome in children and adolescents: Shifting the focus to cardiometabolic risk factor clustering. Pediatrics. 2017;140(2).