ASSOCIATION OF LIPOPROTEIN-α WITH OBESITY IN CHILDREN AND ADOLESCENTS IN DISTRICT SWAT

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Background: Studies have demonstrated that atherosclerosis has its silent beginning during childhood. Coronary artery disease, particularly when it presents early in adult life has been observed to have a familial tendency. Lipoprotein-α [Lp-α], has a strong genetic association and raised levels when combined with obesity increase the risk of premature coronary heart disease. Thus in adults, has emerged the possibility of preventing or delaying the coronary artery disease when appropriate measures are applied early in life. In our study, we assessed the prevalence of overweight and obesity and its association with Lp-α in the child population of district Swat. **Methods:** The study was carried out in Saidu Teaching Hospital and Biochemistry Department, Saidu Medical College, Swat, from May to July 2007. Both boys and girls between 10–20 years of age were included. The prevalence of obesity was detected in 200 subjects by using body mass index. One hundred subjects were selected to compare the anthropometric and cardiovascular parameters of obese subjects with control group. Lp-α was measured in children of both the groups. **Results:** The prevalence of obesity in adolescent boys was 6.7% and that in adolescent girls was 10%. The prevalence was higher in female subjects, compared to male subjects. Obese subjects had significantly higher weight, body mass index, blood pressure values and Lp-α levels as compared to control group. **Conclusion:** Lipoprotein-α level is higher in obese children and adolescents than in non-obese.

Keywords: Lipoprotein-α, Lp-α, Body mass index (BMI), Blood pressure (BP), Obesity

INTRODUCTION

Obesity is defined as a BMI greater than or equals to the 95th percentile for age and gender, overweight as a BMI greater than or equal to the 85th percentile but less than the 95th percentile, and normal weight as BMI less than the 85th percentile.

BMI is recognised as one of the most useful indices for adiposity both in children and adults. BMI is determined by dividing weight (wt) in kilogram by height (ht) in meters squared. It is highly correlated with weight (0.8–0.9). BMI is also highly correlated with body fat (0.7–0.8). Based on data for children 6–15 years, World Health Organization (WHO) has suggested the following cut-off points for BMI as broad guidelines for defining obesity in both genders.[2]

WHO Suggested cut-off point of BMI

BMI threshold	Boys	Girls
Age 10–11	19.0	19.6
Age 12–13	19.4	21.2
Age 14–15	19.6	21.9

Only a small percentage of childhood obesity is associated with a hormonal or genetic defect, with the remainder being idiopathic in nature. An endogenous cause for obesity can be either suspected or eliminated from the differential diagnosis in virtually all children based on a careful history and physical examination.[3]

Parental obesity is the most important risk factor for childhood obesity. Twin, adoption, and family studies indicated that inheritance is able to account for 25–40% of inter-individual difference in adiposity.[4]

Widespread reports indicate that the prevalence of obesity among children and adolescents has been increasing in recent years, just as it has in adults. A recent report of the initial results of 1999 National Health and Nutrition Examination Survey indicates that prevalence rates have increased even further, to 13% of children aged 6–11 years and 14% of adolescents aged 12–19 years.

Obese children under three years of age without obese parents are at low risk for obesity in adulthood, but among older children, obesity is an increasingly important predictor of adult obesity, regardless of whether the parents are obese. Parental obesity increases the risk of adult obesity among both obese and non-obese children less than 10 years of age.[5]

Multiple studies have demonstrated that atherosclerosis has its silent beginning duringchildhood.[6,7] Coronary artery disease, particularly when it presents early in adult life has been observed to have a familial tendency. This clustering of coronary artery disease is partly explained by the familial aggregation of the traditional risk factors (hyper-lipidemia, hypertension and diabetes mellitus)[8], while unknown or new factors such as fibrinogen[9] and Lipoprotein-α may also contribute. Lp-α is the only major lipid risk factor which remains remarkably constant in an individual. In addition, Lp-α shows a strong heritability.[10] Srinivasan *et al*[11] have postulated that serum levels of Lp-α could be substituted for the knowledge of parental history of premature coronary artery disease in distinguishing patients from controls. This will be particularly useful in identifying children at risk when parents are still quite young.

The objective of this study was to investigate the association of Lipoprotein-α with obesity in the overweight and obese children of district Swat.

SUBJECTS AND METHODS

Two hundred subjects (aged 10–20 years) were randomly selected to detect the prevalence of obesity. Hundred subjects, 50 control (non-obese) and 50 obese (age and sex matched) were selected for the assessment of the relationship of obesity indices with cardiovascular parameters.

Both males and females adolescent between ages 10–20 years were included in the study. Subjects were excluded from study participation that had a medical history of disease other than overweight/ hypertension or were taking any medication known to affect metabolism.

Health Scale (Model ZT-120) was used to measure weight and height. Weight was assessed at 2 different points during interview, and the 2 were averaged for these analyses. It was measured to the nearest 0.5 kg. Participants were advised to wear normal clothing without shoes, socks, and belts. Height was also assessed at 2 different points during interview, and the two readings were averaged for these analyses. It was measured to the nearest 0.1 cm.

Body mass index (BMI) was determined by dividing weight (wt) in kilogram by height (ht) in meters squared (BMI=kg/m^2). Body mass index percentile charts were used to determine obesity in boys and girls.

Blood pressure data was obtained, after at least 5 minutes of rest, with subjects in seated position. A mercury sphygmomanometer (Model SM-300), with an appropriate sized cuff covering two third of the upper arm, was used. The onset of the first tapping sound was taken to indicate the systolic blood pressure, while the point of complete disappearance of the sound (Korotkoff V) was taken to indicate diastolic blood pressure. The mean of three reading was recorded. Age and gender specific percentiles of blood pressure measurements charts were used. After a 12 hour fast, venous blood was drawn between 0730 and 0900 hour. Serum for Lp-α measurement was prepared from additive-free blood samples by centrifugation for 10 minutes at 3000 g. Serum was transferred in aliquots to plastic tubes and stored at 4 ºC until assay within 48 hours. Lipoprotein-α was measured in serum by spectrophotometric method using the kit cod: 1107020 supplied by Spinreact, Spain.

Values were expressed as Mean±SD. Differences between means were compared with an unpaired Student's *t*-test and differences in proportions with the Chi-square test, $p<0.05$ was considered significant.

RESULTS

The prevalence of obesity for both boys and girls with age (10–15 years) was 6.7%, for boys with age (16–20 years) 6.7%, for girls with same age was 10%. As shown, the prevalence of obesity in children and adolescents was the same for both genders till the age of 15 years. In the late adolescents, the prevalence increased with increase in age and still higher in female than male. (Table-1).

Table-1: Prevalence of obesity in boys and girls

Age (Years)	Prevalence of Obesity	
	Male Subjects	Female Subjects
10–15	6.7 %	6.7 %
16–20	6.7 %	10 %

The anthropometric parameters such as weight, height, BMI, and both systolic and diastolic blood pressure of Group 'A' (control) were compared with Group B (obese). The mean weight of Group 'A' was 40±7.27 and that of Group 'B' was 62±8.43 ($p<0.001$, highly significant). The mean height of Group 'A' was 1.45±0.08 and that of Group 'B' was 1.44±0.068. The difference in height of two groups were statistically non-significant. The mean BMI of Group 'A' was 19±1.29 and that of Group 'B' was 29.9±1.41 ($p<0.001$, highly significant).

Systolic and diastolic blood pressure of Group 'A' was also compared with Group 'B'. The mean systolic blood pressure of Group 'A' was 90±11.8 mm Hg and that of Group 'B' was 135±8.3 mm Hg ($p<0.001$, highly significant). The mean diastolic blood pressure of Group 'A' was 60±10.9 mm Hg and that of Group 'B' was 85±6.8 mm Hg ($p<0.001$, highly significant). The mean Lp-α of Group 'A' was 20±1.2 and that of Group 'B' was 31±1.6 ($p<0.05$, significant). (Table-2).

Table-2: Comparison of anthropometric and cardiovascular parameters of control group and obese group

Parameters	Group A Control (n=50)	Group B Obese (n=50)	*p*-Value
Weight	40±7.27	62±8.43	**
Height	1.45±0.08	1.44±0.07	
BMI	19±1.29	29.9±1.41	**
Systolic BP	90±11.8	128±8.3	**
Diastolic BP	60±10 .9	85±6.8	**
Lp-α	11.57±1.2	19 .32±1.6	*

Values given as Mean±SD, *$p<0.05$, **$p<0.001$

DISCUSSION

Obesity is an excess body weight due to fat deposition as compared to set standards of body weight. Prevalence of obesity varies amongst countries depending upon environmental and behavioural changes brought about by economic development, modernization and urbanization. The variation in prevalence of obesity

epidemic in various races and communities of the world may be attributed to heredity, age, gender, and diet, eating patterns, life style and/or behaviour.

Obesity tracks from childhood to adulthood,[12] and is strongly related to hypertension in adults.[13] It is suggested that avoidance of obesity should be emphasized in any discussion of blood pressure control in children. At least 30% of obesity begins in childhood. Conversely 50 to 80% of obese children become obese adults.[14]

The present study showed that obesity was prevalent in both adolescent boys and girls in Swat district. The prevalence of obesity in children and adolescents was the same for both genders till the age of 15 years. In the late adolescents, the prevalence increased with increase in age and still higher in female than male. Also a significant relationship of obesity with both systolic and diastolic blood pressure was found.

Two studies conducted in Chennai, Tamil Nadu, India in 1981 and 1998, showed almost similar results.[15] Ogden *et al*, also observed almost similar findings and reported that persistently elevated blood pressure levels occur about 9 times more frequently among obese children and adolescents (ages 5 to 18) than in non-obese. Obese children and adolescents are reported to be 2.4 times more likely to have high diastolic blood pressure and 4.5 times more likely to have high systolic blood pressure than their non-obese peers.[16]

According to WHO, at least 50% of adults and 20% of children in UK and USA are currently overweight.[17] Roberts *et al*[18], reported that the United States has one of the highest obesity rates in the world and is the first nation to have people who are both impoverished and obese. The obesity epidemic is also increasing in Europe, Asia and throughout the Americas: as the Indian newspaper 'The Tribune' states: Obesity also plagues Middle Eastern countries, with 35 per cent of Egyptians considered obese, a greater proportion than the population in the USA at 20%.[18] Evidence from the National Health and Nutrition Examination survey (NHANES) in the United States and the national study of health and growth in the United Kingdom shows an increasing prevalence of overweight and obesity in young children and adolescents.[19,20]

Studies of obesity in Asian subjects show that generalized obesity is the major determinant of cardiovascular risk in the Chinese and East Asian subjects while central obesity is associated with greater cardio-vascular risk in South Asians.[21] Lipoprotein-α is the only major lipid risk factor which remains remarkably constant in an individual. In addition, lipoprotein-α shows a strong heritability. Serinivasn *et al*[11] have postulated that serum lipoprotein levels could be substituted for the knowledge. The strongest correlation was found with lipoprotein-α and they concluded that lipoprotein-α has probably the strongest genetic determinant.

There is scanty literature available about the correlation of serum Lp-α with childhood obesity. Taimela *et al* [22] have reported a statistically significant correlation of serum Lp-α levels with the level of physical activity in children. Cabrinety *et al*[23] report that Lp-α is higher in obese children. Obesesan *et al*[24] have concluded in their study that body mass index or weight were significant predictors of raised Lp-α levels in black children. We report similar result among obese children in our study. We have found a strong correlation of serum Lp-α levels with obesity in children.

CONCLUSION

Lipoprotein-α level is higher in obese children and adolescents than in non-obese. Obesity is common in the young population of Swat. Children should be screened for Lp-α and dietary measures should be taken as a first step approach to reduce the future risk of premature coronary artery disease and its associated comorbidities.

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