Subclinical atherosclerosis in obese adolescents with normal left ventricular function

Amina M. Abdel-Wahab, MD, *Hoda A. Atwa,* MD, *Azza Z. El-Eraky,* MD, *Mohamed A. El-Aziz,* MBBCh.

ABSTRACT

الأهداف: تقييم مدى تأثير السمنة على تضخم الطبقة الداخلية للشريان السباتي، وكتلة البطين الأيسر لدى الشباب المصابين بالسمنة.

الطريقة: أُجريت هذه الدراسة في قسم العيادات الخارجية بمستشفى قناة السويس الجامعي، الإسماعيلية، مصر، وشملت 52 من الشباب المصابين بالسمنة (متوسط العمر: 14.16±2.64 عاماً)، و52 من الشباب الأصحاء الذين كانوا في مجموعة الشاهد (متوسط العمر: 12±2.3 عاماً). لقد خضعت عينة الدراسة لمجموعة الفحوصات التالية: التاريخ المرضي، وبعض الفحوصات السريرية، والفحوصات المختبرية (مستويات السكر في الدم أثناء الصيام، ومستويات الدهون)، وفحص القلب بالموجات فوق الصوتية للكشف عن تضخم البطين الأيسر وأبعاده. وقد قمنا باللجوء إلى موجات دوبلور فوق الصوتية من أجل الكشف عن تضخم الطبقة الداخلية للشريان السباتي.

النتائج: أشارت نتائج الدراسة إلى أن مجموعة الشباب المصابين بالسمنة كانوا يعانون من ارتفاع واضح في معدل الكولسترول الكلي، وثلاثي الجليسريد، والكولسترول منخفض الكثافة، بالإضافة إلى انخفاض مستويات الكولسترول عالي الكثافة بالمقارنة مع مجموعة الشاهد. كما عانت مجموعة الشباب المصابين بالسمنة من زيادة واضحة في ضغط الدم، وتضخم الطبقة الداخلية للشريان السباتي، وتضخم البطين الأيسر، ومؤشر تضخم البطين الأيسر. وأظهرت الدراسة مدى العلاقة بين مؤشر كتلة الجسم وكلاً من: اختلال مستوى الدهون البروتينية، وارتفاع ضغط الدم، وتضخم الطبقة الداخلية للشريان السباتي، والبطين الأيسر، والجدار الخلفي لعضلة القلب. ولقد كان هناك علاقة كبيرة بين تضخم الطبقة الداخلية للشريان السباتي وكلاً من: زيادة الكولسترول منخفض الكثافة، وانخفاض الكولسترول عالي الكثافة، وزيادة ضغط الدم، وتضخم البطين الأيسر، والجدار الخلفي لعضلة القلب.

خاتمة: لقد أثبتت الدراسة مدى ارتباط السمنة في سن الطفولة والشباب بظهور مرض تصلب الشرايين دون السريري، وبالرغم من عدم إصابة الأطفال الذين شملتهم الدراسة بأي خلل في البطين الأيسر إلا أن مجموعة من التغيرات قد ظهرت في تركيب هذا البطين.

Objectives: To assess the impact of obesity on carotid intima media thickness and left ventricular (LV) mass in obese adolescents.

Methods: The study included 52 obese adolescents (mean age 14.16±2.64 years) and 52 healthy adolescents who served as a control group (mean age 12±2.3 years), who were attended the outpatient clinic at Suez Canal University Hospital, Ismailia, Egypt. The study population was submitted for medical history, clinical examination, laboratory investigations (fasting blood sugar and lipid profile), and echocardiographic examination of LV mass and dimensions. Assessment of carotid intima-media thickness was carried out by using carotid duplex. All children had normal LV function.

Results: Obese adolescents had a significant increase in total cholesterol, triglyceride, LDL-C, and low HDL-C compared to the control group. Also, there was a significant increase in blood pressure, carotid intima media thickness, LV mass, and LV mass index. There was a significant correlation between BMI and dyslipidemia, blood pressure, carotid intima/media thickness, LV mass, and posterior wall thickness. Carotid intima-media thickness had a significant correlation with increased LDL-C and low HDL-C, blood pressure, LV mass, and posterior wall thickness.

Conclusion: Obesity in childhood and adolescents is associated with subclinical atherosclerosis. Although obese children had no LV dysfunction, yet there are LV structure changes.

Saudi Med J 2011; Vol. 32 (9): 919-924

From the Department of Pediatrics (Abdel-Wahab, Atwa, El-Aziz), and the Department of Cardiology (El-Eraky), Faculty of Medicine, Suez Canal University, Ismailia, Egypt.

Received 22nd March 2011. Accepted 18th July 2011.

Address correspondence and reprint request to: Dr. Hoda Atwa, Department of Pediatrics, Faculty of Medicine, Suez Canal University, 111 University Hospital Rd, Postal Code 41522, Ismailia, Egypt. Tel. +20 (64) 3327587. Fax. +20 (64) 3231982. E-mail: atwahoda@gmail.com

Obesity is becoming a global epidemic; a dramatic increase in overweight among children and adolescents during the past 2 decades has been documented. Using the international definitions, at least 10% of the school age children are overweight or obese worldwide.[1] The prevalence of obesity in Egypt was 22% in males and 45.5% in females.[2] In Egypt, 7% of boys and 18% of girls were overweight and 6% of boys and 8% of girls were obese.[3] Over the last 30 years, the prevalence of obesity has nearly tripled for youth 12-19 years of age. A prevalence increased of 5-17.4%.[4] Increasing prevalence of obesity among children and adolescents has serious implications for their health because it is associated with comorbidities during childhood, as well as increased risk of chronic diseases as hypertension, dyslipidemia, metabolic syndrome, insulin resistance, diabetes, polycystic ovary syndrome, sleep apnea, endocrine abnormalities, orthopedic disorders, and psychological problems, and decreased life expectancy.[5] Childhood obesity predisposes to increased left ventricular mass,[6] endothelial dysfunction, carotid intima-media thickening as a marker of early preclinical atherosclerosis, and the development of early aortic and coronary arterial fatty streaks and fibrous plaques.[7]

Methods. This cross-sectional study was conducted in the outpatient clinics of the Suez Canal University Hospital, Ismailia, Egypt. The study population included 52 obese adolescents aged from 10-19 years, males, and females with BMI >95th percentile for age and gender. American Medical Association Expert Committee reported that BMI for age at or above 95th percentile defined as obesity.[8] Control group consists of 52 healthy adolescents' age- and gender- matched. Their body mass index was between 25-75th percentile. They were recruited from the relatives of the patients who attended the outpatients' clinics of the Suez Canal University Hospital. Patients with type I diabetes mellitus, cardiac diseases (congenital heart disease, rheumatic heart disease), secondary hypertension or those with syndromes including obesity were excluded. The study was carried out in accordance with the guidelines of the Helsinki Declaration. Informed consents were obtained from all controls and children parents. The study was approved by the Suez Canal University Research and Ethics Committee. All the study population was subjected to regarding the demographic data and the especial habits of medial importance as: a) Regular exercise: playing sports 3 times weekly b) Over watching TV: more than 4 hours/day. c) Computer over user; more than 4 hours/day, d) Excess carbohydrate eating: such as rice, macaroni over eating, and meticulous clinical examination with emphasis on the main outcome measures. Biometric and anthropometric measurements of blood pressure (BP), height, and weight was made. Height was measured in meters. Body mass index (BMI) values were calculated as weight (in kilograms) divided by height squared (in square meters) and were compared with age standards,[9] Waist circumference is defined as the minimal circumference measured at the navel, and the hip circumference is the widest circumference measured at the hips and buttocks.[10]

Fat % = 1.2 (BMI) + 0.23 (age years) - 1.62[11]

Blood pressure was measured by mercury sphygmomanometers on 3 separate occasions. Systolic and diastolic blood pressures were measured with an appropriate size cuff in the right arm with the subject seated, his or her back supported, feet on the floor and right arm supported, and cubital fossa at heart level. The right arm is preferred in repeated measures of BP for consistency and comparison with standard tables. Systolic blood pressure is determined by the onset of the "tapping" Korotkoff sounds (K1) and the fifth Korotkoff sound (K5), or the disappearance of Korotkoff sounds, as the definition of diastolic blood pressure.[12] Transthoracic echocardiographic (TTE) transthoracic echocardiographic measurements were performed with Hewlett Packard Sonos 1800 phased array system using a 3.0-mHz transducer for 2-dimensional M-mode measurements of the left ventricular mass according to the recommendations of the American Society of Echocardiography.[13] M-mode echocardiography was used to measure left ventricular dimensions and left ventricular wall thickness, allowing for the calculation of left ventricular mass after correction for BSA; left ventricular mass was calculated by the following formula:[14] LV mass (gm) = 1.04 [(LVID + PWT + IVST) 3 - LVID3] Ã- 0.8 + 0.6 g.

Where LVID is the internal dimension, PWT is the posterior wall thickness, IVST is the interventricular septal thickness, 1.04 is the specific gravity of the myocardium, and 0.8 is the correction factor. All measurements are made at end-diastole (at the onset of the R wave) in centimeters. Left ventricular mass index was calculated as left ventricular mass (grams) divided by BMI.

High resolution carotid ultrasonographic studies were performed with a Philips HD11, linear array probe 12 MHz with the subject in supine position. The estimation of cIMT was carried out at the Radiology Department, Suez Canal University Hospital. All the children underwent a CIMT measurement by the same experienced doctor and he used the same equipment. He was blinded to the clinical and laboratory characteristics. The study protocol involved scanning of the far wall of the right and left common carotid arteries in the distal 1.0 cm. The crest at the origin of the bifurcation was used as an anatomical landmark to identify the segment to be visualized. Three measurements of the intima–media thickness were averaged, in order to give the mean common carotid intima-media thickness for each side. The thickness of cIMT is an excellent surrogate marker of cardiovascular risk.[15]

Laboratory investigations. Blood samples were drawn after an overnight fast from both groups of adolescents. Fasting total cholesterol, HDL-cholesterol, and triglyceride concentrations were analyzed using enzymatic methods with the use of Boehringer Manheim reagents with a fully automated analyzer (Hitachgi 917; Hitachi, Tokyo, Japan). LDL-cholesterol was calculated using the Freidewald equation,[16] and fasting blood glucose was analyzed using an enzymatic method.

All the data were collected and were statistically analyzed using SPSS 14 program. Numerical data were expressed as mean ± SD. Non-numerical data were expressed as percentage. The mean was compared using the unpaired Student's t test. P value <0.05 was considered statistically significant. Between-group comparisons were made using ANOVA to analyze differences between cases and controls. Correlations were performed by linear regression analysis.

Results. The study included 52 obese adolescents (mean age 14.16± 2.64 years) and 52 adolescents as a control group (mean age 12± 2.3 years). There was an increased duration of watching television and internet using for more than 4 hours and decreased time of physical activities in obese adolescents compared to the control group. Food habits were different in both groups. Family history of obesity and chronic diseases was more common in obese adolescents (**Table 1**). Body mass index, fat % and systolic and diastolic blood pressure were significantly higher in the obese group. Although fasting blood sugar was in the normal range in both groups, it was higher in the obese adolescents (**Table 1**) also they had a significant increase in total cholesterol, triglyceride, LDL-C, and low HDL-C compared to the control group.

Table 2 shows that carotid intima/media thickness, LV mass, and LV mass index were increased in the obese adolescents. LV dimensions were greater in the obese group and ejection fraction although it was in the normal range on both groups; yet it was lower in the obese one. There was a significant correlation between BMI and dyslipidemia, blood pressure, and carotid intima/media thickness (**Figure 1a**). LV mass and posterior wall thickness (**Figure 1b**). Carotid intima/media thickness had significant correlation with increased LDL-C (**Figure 1c**) and low HDL-C, blood pressure, LV mass (**Figure 1d**) and posterior wall thickness. Obese children with metabolic syndrome had significantly higher cIMT than those without metabolic syndrome $p<0.04$. Left ventricular mass was significantly higher in obese

Table 1 - Clinical characteristics of the obese and control adolescents.

Variables	Obese (n=52) (Mean±SD)	Control (n=52) (Mean±SD)	*P*-values
Age (mean±SD)	14.16±2.6	12±2.3	0.01
Gender (male/female)	30/22	25/27	NS
Physical activity			
Regular exercise	3	20	0.001
Over watching TV	46	22	0.01
Computer user for hours	27	19	0.02
Food habits			
Carbohydrate over eating	47	32	0.01
Junk food	45	25	0.01
Family history			
Obesity	17	9	0.01
Diabetes mellitus	9	5	0.03
Hypertension	18	8	0.01
BMI (kg/m^2) (mean±SD)	39.98±39.36	17.90±1.01	0.001
Fat % (mean±SD)	26.26± 4.35	7.99±1.73	0.001
SBP (mmHg) (mean±SD)	118.80±9.17	109.80±8.68	0.001
DBP (mmHg) (mean±SD)	75.60±6.11	66.40± 6.31	0.001
FBS (mean±SD)	87.56 ± 14.62	82.43 ± 2.39	0.017
Total cholesterol (mean±SD)	172.06±16.75	160.10±6.78	0.008
Triglyceride (mean±SD)	90.30±35.28	61.86±5.20	0.007
LDL-C (mean±SD)	113.41±15.47	92.05±8.91	0.009
HDL-C (mean±SD)	52.58±2.71	57.23±1.60	0.005
Children with MS	16	0	

BMI - body mass index, SBP - systolic blood pressure, DBP - diastolic blood pressure, FBS - fasting blood glucose, LDL-C - Low density lipoprotein cholesterol, HDL-C - high-density lipoprotein cholesterol

Table 2 - Echocardiographic findings and carotid intima media thickness (IMT) of obese and control adolescents.

Variables	Obese (n=52) (mean ±SD)	Control (n=52) (mean ±SD)	*P*-value
LV IDd (cm)	50.08±0.38	44.7±0.20	0.004
LV IDs (cm)	32.1±0.33	24.4±0.20	0.002
IVSd (cm)	8.90±0.12	7.4±0.3	0.008
LV PWd (cm)	8.40±0.20	5.4±0.3	0.006
EF%	64.17±4.13	72.52±1.15	0.003
LVMI (gm/m^2)	62.71±7.24	42.29±5.75	0.003
Right carotid IMT (cm)	0.51±0.10	0.40±0.02	0.001
Left carotid IMT (cm)	0.50±0.02	0.40±0.02	0.001

LV IDd - left ventricular internal dimension in diastole, LV IDs - left ventricular internal dimension in systole, IVSd - interventricular septum in diastole, LV PWd - left ventricular posterior wall in diastole, EF - ejection fraction, LVMI - left ventricular mass index

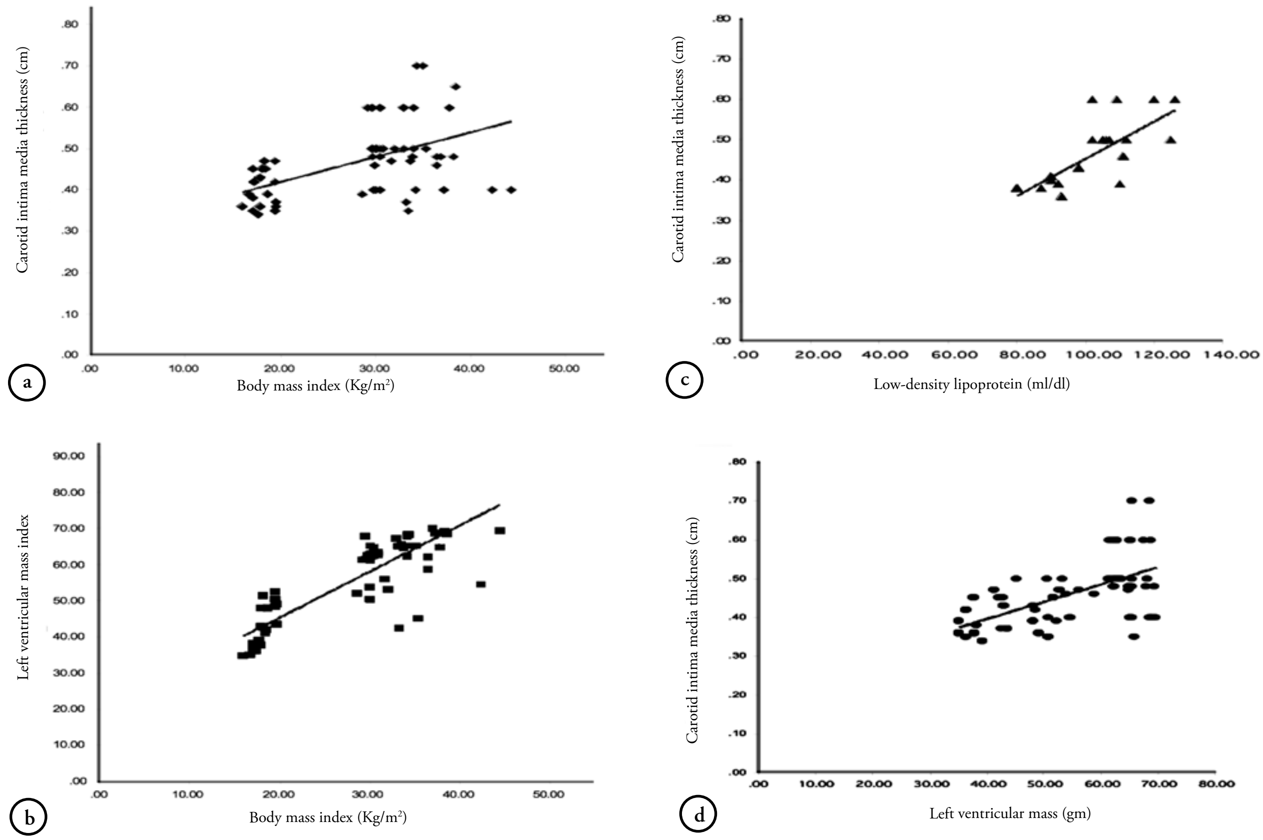

Figure 1 - Correlation between carotid intima media thickness and some related factors. **a**) Correlation between body mass index and carotid intima-media thickness. **b**) Correlation between body mass index and left ventricular mass indes. **c**) Correlation between carotid intima-media thickness and LDL cholesterol level. **d**) Correlation between carotid intima-media thickness and left ventricular mass.

Table 3 - Carotid intima media thickness in obese children with and those without metabolic syndrome.

Obese children	Carotid intima media thickness Mean ±SD	Left ventricular mass Mean ±SD
Children with MS	0.52±0.09	64.9±3.5
Children without MS	0.43±0.15	59.6±4.23
P-value	0.04	0.03

children with metabolic syndrome than those without $p<0.03$ as shown in **Table 3**.

Discussion. Obesity is becoming a global epidemic for both adult and children, and the prevalence of obesity is continuing to rise. It is associated with a significant burden of ill health for obese children and for adults who were obese as children. Pediatric obesity is the leading cause of hypertension, insulin resistance, and dyslipidemia, type 2 diabetes mellitus, and left ventricular hypertrophy and predisposes to endothelial dysfunction, carotid intimal-medial thickness, and increased arterial stiffness.

In this study, the life style was different between the obese adolescents and the control group. Physical activity was lower as they spent more watching television and using computer and internet. Food habits as eating carbohydrates and junk food were more in obese group.[17] They reported that the time spent viewing television has been widely associated with greater adiposity in children. Television viewing could potentially influence adiposity by displacement of physical activity or increase energy intake through snaking promoted by advertising of energy dense food. The family history of obesity, diabetes mellitus and hypertension were more in obese group; this finding is in accordance with the results of Burke.[1] The obese participants had higher blood pressure than normal weight adolescents; these results were similar to that reported by Iannuzzi & Maria,[18] and Wunsch et al.[19] Obesity is strongly associated with higher blood pressure than normal, due to the direct effects of obesity on hemodynamics and the mechanisms linking obesity and an increase in peripheral vascular resistance, endothelial dysfunction, insulin resistance, sympathetic nervous system, substances released from adipocytes (IL-6, TNF-α, and so forth), and sleep apnea.[20] In the present study, obese adolescents had a significant increase in total cholesterol, triglyceride, LDL-C, and low HDL-C compared to the control group. Chinali et al[21] found that obese adolescents had significantly higher values of fasting glucose, lipid profile (higher TG and lower HDL-c). Iannuzzi and Maria[18] showed that obese adolescents had significantly higher plasma concentrations of cholesterol and glucose. Pinhas et al[22] found that the most striking findings in their study were the 5-17 years old obese population, the combination of elevated TG and LDL-c and low HDL-c levels place them at greater cardiovascular risk than their non-obese peers. Carotid intima/media thickness was increased significantly in the obese adolescents. This finding is in accordance with the results of Atabek et al[23] and Wunsch et al.[19] Obesity is associated with abnormal endothelial function due to decrease in nitric oxide, which may be related to an increase in oxidative stress, or may result from pro-inflammatory cytokines. In the Framingham heart study, the BMI was highly associated with systemic oxidative stress, as determined by creatinine- indexed urinary 8-epi-PGF2–α levels.[24] In this study, there is a significant increase in LV mass and LV mass index, LV dimensions were greater in the obese group and ejection fraction although it was in the normal range on both groups; it was lower in the obese group. The same result was demonstrated by Chinali et al[21] and Friberg et al.[25] Increased LVM in overweight and adolescents is strongly associated with increase systolic blood pressure. LVH may be a compensatory response to increased cardiac overload. In obese adolescent, increase in LVM exceeds the need to compensate for increase hemodynamic load.[26] The present study demonstrated a relationship between BMI and cardiovascular risk factors; this suggests that these young obese individuals have a silent risk factor profile. Friberg et al[25] reported in their study that BMI correlated mainly with LV mass and systolic blood pressure, Yang et al[27] reported that BMI correlated with carotid intima /media thickness and blood pressure, also Teixeira et al[28] found in their study that BMI correlated significantly positive with triglycerides and LDL-cholesterol and negatively with HDL-cholesterol. Carotid intima/media thickness had significant correlation with increased LDL-C and low HDL-C, blood pressure, LV mass, and posterior wall thickness that are agreeing with the results of Yang et al[27] and Iannuzzi and Mraia.[18]

Clinical implication. Obesity in childhood and adolescents is associated with increased risk of atherosclerosis and cardiovascular risk factors such as increased carotid intima/media thickness (as a marker of preclinical atherosclerosis), hypertension, dyslipidemia, metabolic syndrome, insulin resistance, diabetes) and decreased life expectancy. Carotid artery duplex scanning is a simple technique to measure carotid intima/media thickness and to diagnose early atherosclerotic changes in obese adolescents.

Some limitations exist in the present study. These limitations include the limited number of patients recruited within the study. There are no intima/media thickness normal values for Egyptian children and adolescents, which need a study of a large number of this age group to detect the normal values.

In conclusion, obesity in childhood and adolescents is associated with subclinical atherosclerosis. Although obese children had no LV dysfunction, yet there are left ventricular structural changes.

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Related topics

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