# ‘Infectobesity’ in egyptian adolescent women and its relations to carotid intima–media thickness

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**Background**
‘Infectobesity’ is a new term to describe obesity of infectious origin, such as infection by human adenovirus-36 (Adv36). It appears to be a new concept, evolved over the past 20 years. Visceral obesity is associated with a higher risk of cardiovascular disease. Increased carotid intima–media thickness (CIMT), a marker of early-onset atherosclerosis, has been observed in obese children and adolescents. The present study aims to investigate the relationship between visceral obesity, CIMT, and Adv36 in female Egyptian adolescents.
**Patients and methods**
The present study included 90 women aged 12–15 years. It was conducted at the Medical Excellence Research Center of the National Research Centre, Cairo, Egypt, during the period between September 2016 and November 2017. Anthropometric assessment was done. Fasting blood samples were withdrawn for the measurement of Qualitative Human Adv36 antibody using a sandwich enzyme-linked immunosorbent assay. Fasting plasma glucose was determined calorimetrically, by the glucose oxidase method and insulin level using the solid-phase enzyme-linked immunosorbent assay and lipid profile. Visceral obesity was measured by an abdominal ultrasound. CIMT for both carotid arteries were measured by high-resolution echo Doppler.
**Results**
Girls with visceral obesity ($n$=26) had higher frequency of increased CIMT at left (96.2 vs. 75%), right carotid artery (84.6 vs. 73.4%) and Adv36 sero-positive antibody (69.2 vs. 56.2%) than among those without visceral obesity ($n$=64). Among the total samples, visceral obesity had significant positive correlations with BMI, waist and hip circumference, while it had insignificant correlations with age, blood pressure (BP), CIMT at right and left carotid arteries, adenovirus and laboratory findings. CIMT had a significant positive correlation with each other, insulin resistance and total cholesterol, and significant negative correlations with high-density lipoprotein and waist circumference. Adv36 had significant negative correlations with BP (both systolic and diastolic) and significant positive correlation with insulin level. Adv36 and CIMT had insignificant correlations with each other and with the anthropometric measurements, BP, visceral obesity, triglycerides, and low density lipoprotein.
**Conclusion**
The frequency of Adv36 and increased CIMT at left carotid artery were higher among girls with visceral obesity than among those without visceral obesity. However, visceral obesity, CIMT at both right and left carotid arteries, and Adv36 had insignificant correlations with each other.

**Keywords:**
adenovirus 36, adolescent girls, carotid intima, media thickness, visceral obesity

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## Introduction

‘Infectobesity’, the new term that has evolved over the last 30 years, refers to obesity caused by infectious agents. Among 10 different pathogens caused obesity in mice, chicken, and in nonhuman organisms, only adenovirus 36 (Adv36) has been clearly related to cause human obesity [1].

Adenoviruses are considered one of the most popular viral infections in early childhood, responsible for nearly one-third of upper respiratory tract infections; which leads to mild and mostly self-limiting diseases [2]. Adv36 is the only one of the human adenoviruses that was found to have correlations with obesity in human beings [3].

Obesity in the adolescence period of life has major importance not only for the affected adolescents, but also for their society. Adolescents who has obesity, specially visceral, during this period of their life usually will have obesity tracking into the adulthood, which leads to many medical health problems resulting in early death [4]. Vijayakumar *et al.* [5] have described the secular trends in children's and adolescent's physical growth, and reported an increased prevalence of overweight/obese in all age groups and in both sexes.

Visceral fat accumulation causes a lot of metabolic disorders like insulin resistance (IR) and hypertension, through decreasing levels of adipocytokines which lead to cardiovascular risks and premature death due to early atherosclerosis [6]. However, subclinical atherosclerosis starts in early childhood, then progress into the adult life [7].

Increased carotid intima–media thickness (CIMT) and aortic stiffness, as markers of arterial atherosclerosis, have been greatly related (in both children and adults) to the occurrence of cardiovascular risk factors [8]. CIMT is a mirror reflecting an image of remodeling and smooth muscle cell hypertrophy due to increased blood pressures (BPs), one of the most important cardiovascular risk factor [9].

So, the aim of the current study was to investigate the relationship between visceral obesity, CIMT, and Adv36 among Egyptian adolescent women.

## Patients and methods

### Patients

This cross-sectional study included 90 adolescent girls in an age range of 12–15 years. It was conducted at the 'Medical Excellence Research Center' of the 'National Research Centre', during the period between September 2016 and November 2017.

Every girl included in the study was evaluated by the following methods: a full history taking, thorough clinical general and local examination, anthropometric assessment, abdominal ultrasound (US), and carotid artery US and laboratory investigations.

### Ethical approvals

The ethical approvals were obtained from both the Ethics Committee of 'Faculty of Postgraduate Childhood Studies' and from the 'National Research Centre' (approval no.15089). A verbal approval was taken from every girl participated in the current study, in addition to a written informed consent from one of her parents, after explanation of the aim of the study, as well as its possible benefits in avoiding the hazardous health effects of obesity.

### Methods

The full history was taken from apparently healthy participants. It included presence of any present disease and past family history [history of obesity, previous infection, hypertension, cardiovascular diseases (CVD), and diabetes].

Thorough clinical general and local examination was done to exclude organic or genetic disorders that might interfere with the individual's normal growth.

#### *Blood pressure measurements*

Triplicate BP measurements were performed in the sitting posture after 5 min rest and with at least 1 min between recordings, using validated (in pediatric population) semiautomated devices (Omron 705IT). An appropriate cuff was used according to the individual's arm circumference (inflatable bladder size, 13×23, or 15×30 $cm^2$ were appropriate). The average of the last two measurements was used in the analysis. BP was evaluated according to age and sex. A participant was considered hypertensive if her systolic blood pressure (SBP) and/or diastolic blood pressure (DBP) was at least 90th percentile for age and sex. Then according to her BP, the participants were classified into normal (120/80 mmHg), prehypertensive (125/85 mmHg), and hypertensive (130/85 mmHg), according to Lurbe *et al.* [10].

#### *Anthropometric measurements*

For every participant girl, the following anthropometric measurements were taken: body weight, height, waist circumference (WC), and hip circumference (HC). Then, BMI, waist/height ratio, and waist/hip ratio were calculated. All measurements were taken by a well-trained researcher and her assistant, using standardized equipment and following the recommendations of the International Biological Program, and then the mean of three consecutive measurements was recorded [11].

Body height was measured to the nearest 0.1 cm using a Holtain portable anthropometer (The Harpenden Portable Stadiometer, Wales, UK). Body weight was determined to the nearest 0.01 kg using the Seca scale (Seca Balance Beam Scale Model 700, Seca Deutschland Medical Scales and Measuring Systems; Seca GmbH and Co., Hamburg, Germany), with the individual dressed in minimum clothes and no shoes.

BMI was calculated as weight (in kg) divided by height (in m) squared. WC was measured at the level of the umbilicus with the girl standing and breathing normally. HC was measured, while the participant girl was wearing light clothing; at the widest level over the greater trochanters in a standing position and by the same examiner. Circumferences were measured, using nonstretchable plastic tape, to the nearest 0.1 cm. The normal WC value for women is 88 cm=35 inches [12].

*Abdominal ultrasound*

US examination, to every girl, was done to evaluate visceral fat at the umbilicus ultra sound visceral fat (USVF) in cm. Intra-abdominal fat thickness measurement was obtained using the 'Medison SonoAce X8' Ultrasonographic equipment. For the visceral fat, a 3.5 MHz transducer was transversely positioned 1 cm above the umbilical scar on the abdominal midline, without exerting any pressure over the abdomen. The visceral fat thickness attempted corresponding to the measurement in centimeters between the internal surface of the abdominal rectus muscle and the posterior aortic wall in the abdominal midline, during expiration. Subcutaneous fat (S) – distance from the skin to the linea alba, measured on the hemisterna line, 1 cm above the umbilical scar, utilizing the linear transducer in a longitudinal section. Umbilical fat normal value is 4.47 cm, and girls with higher values were considered to have visceral obesity [13].

*Carotid artery ultrasonography*

This examination was performed using a high-resolution echo-Doppler device with a 7 MHz linear transducer. All participants in the study were examined in the supine position, with the head overextended and turned 45° away from the examined side. Both carotid arteries were visualized longitudinally, so that the CIMT of their distal wall was apparent. The best images of the distal wall were used to calculate the CIMT of the common and internal carotid arteries. The value of the CIMT was defined as the mean value of measurements between the right and left carotid arteries, calculated from 10 measurements on each side, 10 mm from the bifurcation of the common carotids [14].

Among women aged less than 30 years, CIMT values of at least 75th per centile were considered high and indicative of increased CVD risk, whereas values between 25th and 75th percentile were considered average and indicative of unchanged CVD risk. Values up to 25th percentile were considered lower CVD risk [15]:

(1) Cutoff point for right common carotid artery=0.39–0.43 (25th to 75th percentile).
(2) Cutoff point for left common carotid artery=0.30–0.47 (25th to 75th percentile).

**Laboratory investigations**

Venous blood samples (5 ml) were obtained in the morning by venipuncture after 12-h overnight fasting. The blood samples were left to clot, and then sera were separated by centrifugation for ~20 min at 1000*g* (or 3000 rpm) within 30 min after collection. Serum was then divided into two parts, one part of them stored at −80°C for Adv36 and insulin tests and the other part was tested for fasting glucose and lipid profile tests.

Qualitative human Adv36 antibody was measured using a sandwich enzyme-linked immunosorbent assay (ELISA), to qualitatively analyze human Adv36 antibody [16] using ELISA kit of MyBioSource Inc. (USA).

Serum insulin was measured using ELISA immunoassay kit of Immunospec Corporation (Livonia, Michigan, USA), according to the method of Kurtoglu *et al.* [17].

Fasting blood glucose was determined calorimetrically, by the glucose oxidase method [18]. Homeostasis model assessment for insulin resistance (HOMA-IR) was calculated as [fasting glucose (mg/dl)×fasting insulin (lU/ml)/405], according to Shashaj *et al.* [19].

Plasma total cholesterol (TC) level [20], triglycerides (TG) [21], and high-density lipoprotein cholesterol (HDL-C) [22] were measured using commercially available kits provided by Stanbio Laboratory Inc. (Boerne, Texas, USA). Low-density lipoprotein cholesterol (LDL-C) was calculated according to the equation developed by Friedewald *et al.* [23] as follows:

$$\mathrm{LDL-C} = \text{total cholesteroltriglycerides}/5 + \mathrm{HDL-C}.$$

**Statistical analysis**

It was performed using the computer program statistical package software for Windows, version 16 (SSPS Inc., Chicago, Illinois, USA). Visceral fat; at umbilicus; cutoff point of 4.47 cm was used to classify the girls under study into two groups: those above

4.47 cm were considered to have visceral obesity, while those with up to 4.47 cm were considered without visceral obesity. Descriptive statistics (mean±SD) was calculated for the anthropometric and laboratory assessment and the ultrasound findings. Student's *t*-test was used to compare the two groups. Frequency distribution of the high-risk groups was presented as number and percentage. $\chi^2$-Test was used to compare the qualitative data. Pearson's correlation was used to assess the association between CIMT and anthropometric measurements, as well as the laboratory findings (quantitative data). Spearman's correlation was used to assess the association between adenovirus (qualitative data) and other variables. Standards of probability were set to *P* value of less than 0.01; which is considered highly significant and *P* value of less than 0.05 which is considered statistically significant, in all analyses.

## Results

The participants under investigation were classified into two group: girls with ($n$=26) or without ($n$=64) visceral obesity.

Adv36 was detected among 56.2% (36/64) of girls without visceral obesity versus 69.2% (18/26) of those with visceral obesity, revealing significant differences between the two groups using the $\chi^2$-test (Fig. 1).

Girls with visceral obesity had the highest significant values for most of the anthropometric parameters (weight, BMI, WC, HC), visceral fat at umbilicus ($P$<0.05), and the lowest significant value in HDL. There were insignificant differences between the two groups in SBP, DBP, CIMT at right and left carotid, and for most of the laboratory investigations (fasting blood glucose, insulin, HOMA, TC, TG, and LDL) (Table 1).

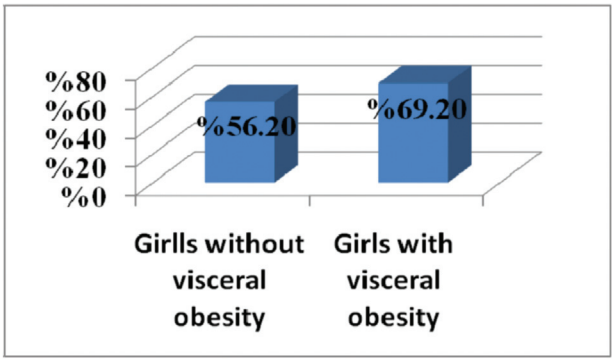
Moreover, girls with visceral obesity had highly significant higher frequency of wide WC (76.9 vs. 34.4%), significantly higher frequency of decreased HDL (50 vs. 37.5%) and increased CIMT at left carotid artery (96.2 vs.75%) and insignificant higher frequency of hypertension; both DBP (34.6 vs. 31.2%) and SBP (19.2 vs. 7.8%), and SBP prehypertension (38.5 vs. 25%), and increased right CIMT (84.6 vs. 73.4%), increased plasma insulin (30.8 vs. 20.3%), HOMA-IR (46.2 vs. 40.6%) and fasting blood glucose (19.2 vs. 6.2%) than those without visceral obesity. While girls without visceral obesity had significantly higher frequency of increased LDL (26.6 vs. 7.7%) and insignificantly higher frequency of increased plasma TC (6.2 vs. 3.8%) and TG (7.8 vs. 3.8%; Table 2).

Visceral obesity at umbilicus, among the total sample, had significant positive correlations with body weight, BMI, WC, and HC, while it had insignificant correlations with age, BP, body height, CIMT at both right and left carotid arteries, laboratory finding and adenovirus (Tables 3 and 4).

Adenovirus had significant negative correlations with BP both SBP and DBP; among total sample and among girls with visceral obesity; and with fasting blood glucose among girls with visceral obesity. It had significant positive correlation with insulin level among the total samples. It had insignificant correlations with age, anthropometric measurements, visceral obesity at umbilicus, and CIMT at both right and left carotid arteries, HOMA, cholesterol, TG, HDL, and LDL (Tables 3 and 4).

CIMT at the left carotid artery had significant positive correlation with CIMT at right carotid artery, HOMA-IR among total sample and among girls with visceral obesity, and TC among the total samples only. It had significant negative correlations with HDL and WC among the total samples only. CIMT had insignificant correlations with the other anthropometric measurements such as age, BP, visceral obesity at umbilicus, insulin, glucose, TG, and LDL (Tables 3 and 4).

**Figure 1**




Frequency distribution of the adenovirus regarding girls with or without visceral obesity.

## Discussion

Measurement of CIMT, which is a noninvasive, feasible, reliable, and inexpensive tool to diagnose early vascular damage, is considered as a marker for

**Table 1 Characteristics, anthropometric, radiological, and biochemical results of the girls with and without visceral obesity**

| Variables | Girls without visceral obesity (*N*=64) (mean±SD) | Girls with visceral obesity (*N*=26) (mean±SD) |
|---|---|---|
| Age (years) | 13.027±1.5448 | 13.683±1.283 |
| SBP (mmHg) | 121.97±9.004 | 125.5±9.331 |
| DBP (mmHg) | 80.45±11.204 | 83.46±11.293 |
| Anthropometry | | |
| Weight (kg) | 62.35±18.02 | 73.45±20.81* |
| Height (cm) | 152.06±9.37 | 154.19±9.08 |
| BMI (kg/m$^2$) | 26.21±6.14 | 30.47±6.78* |
| Waist circumference (cm) | 80.30±12.98 | 91.69±10.94** |
| Hip circumference (cm) | 98.33±16.55 | 107.08±15.94* |
| Radiology | | |
| Umbilical (cm) | 3.02±0.60 | 5.32±0.87** |
| Right CIMT (mm) | 0.05±0.01 | 0.05±0.01 |
| Left CIMT (mm) | 0.05±0.01 | 0.05±0.00 |
| Laboratory | | |
| Insulin (mU/l) | 17.76±14.20 | 19.16±19.62 |
| HOMA | 3.66±3.22 | 3.96±3.68 |
| Glucose (mg/dl) | 84.81±28.94 | 99.45±75.31 |
| Cholesterol (mg/dl) | 166.95±30.70 | 156.65±35.20 |
| TG (mg/dl) | 96.00±46.58 | 103.90±37.71 |
| HDL (mg/dl) | 43.71±7.93 | 37.74±9.11** |
| LDL (mg/dl) | 104.04±26.80 | 98.13±35.45 |

CIMT, carotid intima–media thickness; DBP, diastolic blood pressure; HDL, high-density lipoprotein; HOMA, homeostasis model assessment; LDL, low-density lipoprotein cholesterol; SBP, systolic blood pressure; TG, triglycerides. *$P>0.05$, significant differences using Student's *t*-test. **$P>0.01$, highly significant differences using Student's *t*-test.

**Table 2 Frequency distribution for the high risk among girls with and without visceral obesity**

| Variables | Girls without visceral obesity (*N*=64) [*n* (%)] | Girls with visceral obesity (*N*=26) [*n* (%)] | *P* |
|---|---|---|---|
| SBP (prehypertensive) (120–130 mmHg) | 16 (25.0) | 10 (38.5) | 0.085 |
| SBP (hypertensive) (<130 mmHg) | 5 (7.8) | 5 (19.2) | 0.529 |
| DBP (hypertensive) (85 mmHg) | 20 (31.2) | 9 (34.6) | 0.757 |
| Anthropometry | | | |
| Large WC<88 cm | 22 (34.4) | 20 (76.9) | 0.000** |
| Radiology | | | |
| Right carotid high risk<0.043 mm | 47 (73.4) | 22 (84.6) | 0.256 |
| Left carotid high risk<0.043 mm | 48 (75.0) | 25 (96.2) | 0.020* |
| Laboratory | | | |
| Insulin high risk<30 mU/l | 13 (20.3) | 8 (30.8) | 0.290 |
| HOMA high risk=3.16 | 26 (40.6) | 12 (46.2) | 0.642 |
| Glucose high risk<100 mg/dl | 4 (6.2) | 5 (19.2) | 0.063 |
| Cholesterol high risk<200 mg/dl | 4 (6.2) | 1 (3.8) | 0.652 |
| TG high risk<150 mg/dl | 5 (7.8) | 1 (3.8) | 0.484 |
| HDL high risk<39.9 mg/dl | 24 (37.5) | 13 (50.0) | 0.027* |
| LDL high risk>130 mg/dl | 17 (26.6) | 2 (7.7) | 0.047* |

DBP, diastolic blood pressure; HDL, high-density lipoprotein; HOMA, homeostasis model assessment; LDL, low-density lipoprotein cholesterol; SBP, systolic blood pressure; TG, triglycerides; WC, waist circumference. *$P<0.05$, significant differences using $\chi^2$-test. **$P<0.01$, highly significant differences using $\chi^2$-test.

increased cardiovascular risk and development of subclinical atherosclerosis in adults, as well as in children [24].

In the current study, girls with visceral obesity had the highest significant values in BMI, WC, HC, and visceral fat at the umbilicus, compared with those without visceral obesity. Moreover, girls with visceral obesity had highly significantly higher frequency of wide WC, and insignificantly higher frequency of hypertension, both DBP and SBP, SBP prehypertension, than those without visceral obesity. This agrees with the studies of Al-Hazzaa *et al.* [25] in Saudi Arabia; Kankana *et al.* [26] in India; and El-

**Table 3 Correlation between visceral obesity at umbilicus, adenovirus-36, and carotid intima–media thickness at left carotid artery with the clinical variable**

| Variables | Visceral obesity[a] | | Adenovirus-36[b] | | Left CIMT[a] | |
|---|---|---|---|---|---|---|
| | *r* | *P* | *r* | *P* | *r* | *P* |
| SBP (mmHg) | 0.16 | 0.13 | –0.383 | 0.00** | 0.075 | 0.485 |
| DBP (mmHg) | 0.13 | 0.22 | –0.243 | 0.02* | 0.167 | 0.115 |
| Anthropometry | | | | | | |
| Weight (kg) | 0.258 | 0.01* | –0.086 | 0.42 | 0.001 | 0.995 |
| Height (cm) | 0.04 | 0.69 | –0.067 | 0.53 | –0.014 | 0.895 |
| BMI (kg/m$^2$) | 0.327 | 0.00** | –0.024 | 0.82 | –0.006 | 0.953 |
| Waist circumference (cm) | 0.417 | 0.00** | –0.096 | 0.37 | –0.213 | 0.044* |
| Hip circumference (cm) | 0.252 | 0.02* | –0.089 | 0.41 | –0.024 | 0.826 |
| Radiology | | | | | | |
| Umbilical fat (cm) | | | 0.081 | 0.451 | –0.067 | 0.527 |
| Right CIMT (mm) | –0.075 | 0.485 | 0.029 | 0.789 | 0.937 | 0.000** |
| Left CIMT (mm) | –0.067 | 0.527 | 0.059 | 0.583 | | |
| Laboratory | | | | | | |
| Insulin (mU/l) | –0.099 | 0.385 | 0.269 | 0.017* | 0.217 | 0.054 |
| HOMA | –0.082 | 0.473 | 0.207 | 0.067 | 0.237 | 0.035** |
| Glucose (mg/dl) | 0.093 | 0.386 | –0.038 | 0.727 | –0.033 | 0.755 |
| Cholesterol (mg/dl) | –0.016 | 0.883 | 0.104 | 0.335 | –0.266 | 0.011* |
| TG (mg/dl) | 0.039 | 0.716 | 0.034 | 0.756 | –0.075 | 0.480 |
| HDL (mg/dl) | –0.164 | 0.123 | 0.029 | 0.785 | –0.328 | 0.002** |
| LDL (mg/dl) | 0.019 | 0.857 | 0.156 | 0.146 | –0.171 | 0.106 |
| Adenovirus-36 | 0.121 | 0.262 | | | 0.027 | 0.806 |

CIMT, carotid intima–media thickness; DBP, diastolic blood pressure; HDL, high-density lipoprotein; HOMA, homeostasis model assessment; LDL, low-density lipoprotein cholesterol; SBP, systolic blood pressure; TG, triglycerides. *$P<0.05$, significant correlation using Pearson's a or Spearman's b. **$P<0.01$, highly significant correlation using Pearson's a or Spearman's b.

**Table 4 Correlation between visceral obesity at umbilicus, adenovirus-36, and carotid intima–media thickness at left carotid artery with the clinical variable among girls with visceral obesity**

| Variables | Adenovirus[b] | | Left CIMT[a] | |
|---|---|---|---|---|
| | *r* | *P* | *r* | *P* |
| SBP (mmHg) | –0.627 | 0.001** | –0.296 | 0.143 |
| DBP (mmHg) | –0.466 | 0.016* | –0.182 | 0.374 |
| Anthropometry | | | | |
| Weight (kg) | –0.234 | 0.249 | –0.285 | 0.159 |
| Height (cm) | –0.364 | 0.068 | –0.435 | 0.027* |
| BMI (kg/m$^2$) | 0.201 | 0.325 | –0.135 | 0.509 |
| Waist circumference (cm) | –0.387 | 0.051 | –0.305 | 0.129 |
| Hip circumference (cm) | –0.135 | 0.511 | –0.104 | 0.612 |
| Radiology | | | | |
| Umbilical fat (cm) | 0.100 | 0.626 | –0.239 | 0.239 |
| Right CIMT (mm) | 0.102 | 0.618 | 0.782 | 0.000** |
| Left CIMT (mm) | 0.242 | 0.233 | | |
| Laboratory | | | | |
| Insulin (mU/l) | 0.277 | 0.201 | 0.383 | 0.072 |
| HOMA | – 0.166 | 0.449 | 0.436 | 0.038* |
| Glucose (mg/dl) | –0.660 | 0.000** | –0.097 | 0.638 |
| Cholesterol (mg/dl) | –0.090 | 0.664 | 0.072 | 0.727 |
| TG (mg/dl) | 0.145 | 0.478 | 0.359 | 0.072 |
| HDL (mg/dl) | 0.062 | 0.765 | –0.235 | 0.247 |
| LDL (mg/dl) | –0.134 | 0.513 | 0.056 | 0.787 |
| Adenovirus-36 | | | 0.242 | 0.233 |

CIMT, carotid intima–media thickness; DBP, diastolic blood pressure; HDL, high-density lipoprotein; HOMA, homeostasis model assessment; LDL, low-density lipoprotein cholesterol; SBP, systolic blood pressure; TG, triglycerides. *$P<0.05$, significant correlation using Pearson's a or Spearman's b. **$P<0.01$, highly significant correlation using Pearson's a or Spearman's b.

Kassas and Ziade [27] in Lebanon; who found that adolescent girls with visceral obesity had higher BMI, HC, and wide WC. McGrowder [28] in Brazil and Manios *et al.* [29] in Greece have reported that visceral obese adolescents had a significant increase in both SBP and DBP than normal-weight adolescents. Wyszyńska *et al.* [30] studied 568 adolescents with mild intellectual disabilities and assessed visceral obesity by WC, observed that increased risk of hypertension was more than three-fold higher in adolescents with visceral obesity than in participants with normal WC. Also, the hypertension values were increased stridently in girls with WC of at least 90 percentile.

In the current study, girls with visceral obesity had significantly higher frequency of decreased HDL than those without visceral obesity, while girls without visceral obesity had significantly higher frequency of increased LDL. In agreement with the current results, Kankana *et al.* [26] found that visceral obese adolescent girls had increased risk to have chronic heart disease later on in the future. Sato *et al.* [6] in Japan reported that participants, from both sexes, with visceral obesity had significantly low HDL-C; but in contrast to current results, they had higher serum TG level and higher fasting blood glucose level than patients without visceral obesity, and with no effect on serum LDL-C.

The present study revealed that girls with visceral obesity had significantly higher risk of increased CIMT at the left carotid artery, but insignificant at the right one than in girls without visceral obesity. In agreement with the current study, Baroncini *et al.* [31] in Brazil observed that CIMT is constant in healthy children at age less than 10 years, regardless of sex or BMI, while CIMT increased after that age in all the study samples. Turer *et al.* [32] studied 2893 adolescents aged 12–18 year, from four studies (Cardiovascular Risk in Young Finns, Childhood Determinants of Adult Health, Bogalusa Heart, and Insulin Studies). They reported that obesity is an important risk factor which was strongly associated with increased CIMT. Gooty *et al.* [7] observed that the association between obesity and increased CIMT was stronger in adolescents than in young children. In the study of Atherosclerosis Risk in Young Adults in the Netherlands, Eikendal *et al.* [9] studied adolescents and young adults where the mean age of adolescents was 13.5 years and young adults was 28.4 years. They found that obese adolescents with high SBP had experienced increased CIMT in their young adulthood life. Kollias *et al.* [33] studied 448 children and adolescents from both sexes (aged 10–18 years) from a semirural area in Greece, and found that visceral adiposity, as well as SBP was related to increased CIMT values in healthy adolescents. The CIMT values in the left side were higher than on the right side, thus defining the cardiovascular risk (SBP).

The current study revealed that visceral obesity at umbilicus had insignificant correlations with BP, CIMT at both right and left carotid arteries, and Adv36. Reviewing literature, we found that the relations between visceral obesity, CIMT, and Adv36 were not discussed before. In concordance with the current results, Berger *et al.* [34] found no association between Adv36 and visceral obesity.

The present study showed that CIMT at left carotid artery had significant positive correlation with CIMT at right carotid artery, HOMA-IR and TC, and significant negative correlations with HDL. It had insignificant correlations with other anthropometric measurements, BP visceral obesity at umbilicus, insulin, glucose, TG, and LDL. In agreement with the current study, Epifanio *et al.* [35] in Brazil reported that CIMT on the right side was positively correlated with HOMA-IR, but had insignificant correlations with TG, LDL, glucose, insulin, and HDL. Silva *et al.* [36] in Brazil observed that CIMT was positively correlated with HOMA-IR, a weak positive correlation with LDL, and was inversely correlated with HDL in adolescents. Ryder *et al.* [37] in Minnesota found that CIMT was significantly positively related to IR. Kupfer *et al.* [38] in Brazil also found that there was insignificant correlation between CIMT and TG and LDL. Önal *et al.* [39] found an insignificant correlation between serum triglyceride, LDL, and CIMT. Gao *et al.* [40] in Cincinnati studied 784 African-American adolescents aged 10–24 years and they found that CIMT was negatively correlated with HDL, but positively correlated with cholesterol. In disagreement with our results, they found positive correlations between CIMT with SBP and DBP, LDL, TG, and fasting glucose. Silva *et al.* [36] in Brazil observed that CIMT was positively correlated with BMI and WC among adolescents. Ryder *et al.* [37] in Minnesota found that CIMT was significantly positively related to visceral obesity. Önal *et al.* [39] found insignificant correlation between serum cholesterol and CIMTs. Vijayakumar *et al.* [5] found that CIMT was positively correlated with visceral obesity and WC as one of the important abdominal fat indices. Ge *et al.* [41] in South Asians found that the WC was correlated to CIMT. Rumińska *et al.* [42] showed no correlation between

CIMT and IR. Kupfer *et al.* [38] in Brazil found insignificant correlation between CIMT and WC, cholesterol, and HDL.

The present study showed that the frequency of Adv36 was significantly higher among girls with visceral obesity (69.2%) than among girls without visceral obesity (56.2%). Our results agree with many studies, for example, Parra-Rojas *et al.* [43] in Mexico and Cakmakliogullari *et al.* [44], Karamese *et al.* [45], and Kocazeybek *et al.* [46] in Turkey. They found significantly higher frequency of Adv36 sero-positive antibody among obese adolescents than normal peers. In a study in USA, Broderick *et al.* [47] studied US military personnel, and they found a significant association between Adv36 positivity and female sex; and attributed this to estrogens that may play an important role in increasing the susceptibility to Adv36 infection. Moreover, Atkinson *et al.* [48] and Gabbert *et al.* [49] found a significantly greater WC as a marker of visceral obesity in Adv36-positive obese Italian children, although Na *et al.* [50] did not find the same relation.

In this study, Adv36 had significant negative correlations with BP both SBP and DBP and fasting blood glucose, and significant positive correlation with insulin level, but it had insignificant correlations with WC, HC, visceral obesity at umbilicus, and CIMT at both right and left carotid arteries, HOMA, cholesterol, TG, HDL, and LDL. This coincided with the study of Rogers *et al.* [51] which found that human Adv36 infections in adult White women was associated with significantly lower fasting glucose levels; in addition, Adv36 proteins may provide novel therapeutic targets for the treatment of diabetes mellitus. Ergin *et al.* [52] and Karamese *et al.* [45] in Turkey found an insignificant difference in cholesterol and triglyceride levels between the Adv36-positive antibodies patients and normal ones. Yamada *et al.* [53] found that sero-positive patients (adults and children) with Adv36 are associated with the risk of obesity and increased body weight, with no association with visceral obesity (WC). Na *et al.* [50] and Aldhoon-Hainerova *et al.* [54] did not find a correlation between visceral obesity and Adv36-positive antibodies. Almgren *et al.* [55] found that blood lipid profile levels (TG, TC, and LDL-C) did not differ between Adv36-positive and Adv36-negative Swedish overweight/obese children.In contrast to our results, Parra-Rojas *et al.* [43] found that adolescents with Adv36-positive serum antibodies had higher TC, TG, LDL-C, HOMA, and lower HDL-C levels than in the Adv36-negative group. Trovato *et al.* [56] reported that women with sero-positive Adv36 antibodies had positive correlation with BMI, DBP, HOMA, and TG, while having negative correlation with HDL cholesterol. Na *et al.* [50] in Korea found that obese Adv36-positive children had significantly increased levels of TG, TC, and LDL-C related to Adv36-negative children. In a cross-sectional study, Zarkesh *et al.* [57] found that infection with human Adv36 had positive correlation with TG, TC, LDL-C, and SBP and lower HDL-C levels. Atkinson *et al.* [48] reported a reduction in lipid levels in the presence of Adv36 infection. They demonstrated that Adv36 induced an obese state while paradoxically the serum levels of TG and cholesterol will reduce significantly. Sohrab *et al.* [58] found that Adv36 sero-positive populations had a reduction of total serum cholesterol and TG which in turn increased their body weight and shifting of HDL to LDL cholesterol. Adv36 affected the enzymatic functions mediated in the lipid and glucose uptake. It also reduced the expression and secretion of leptin leading to an increase in appetite, and TG accumulation of fat tissue.

In agreement with our study, a meta-analysis of 10 studies was done from different countries all over the world. This included those of Na *et al.* [50] in Korea and Yamada *et al.* [53], who reported that the risk of obesity and overweight was associated with infection by human Adv36, although being not associated with waist circumstance, suggesting that virus infection is generally related to obesity rather than visceral obesity.

## Conclusion

The frequency of Adv36 and increased CIMT at left carotid artery were higher among girls with visceral obesity than among those without visceral obesity. However, visceral obesity, CIMT at both right and left carotid arteries, and Adv36 had insignificant correlations with each other.

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H. Abdelrahman was responsible for laboratory investigations. Mohamed Kh. Metkees was responsible for radiological examination. Ayman Nada shared in drafting the article. Amany Ebrahim and Walaa Saad collected the data. All authors contributed to the collection of references, drafting of the article, and final approval of the version to be submitted. All the authors have accepted responsibility for the entire content of this submitted manuscript and approved submission.

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There are no conflicts of interest.

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