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Abdominal Obesity and Metabolic Syndrome Burden in Adolescents-Penn State Children Cohort Study

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Abstract

INTRODUCTION—To investigate the association between abdominal obesity and metabolic syndrome (MetS) burden in a population-based sample of adolescents.

METHODS—We used the data from 421 adolescents who completed the follow-up examination in the Penn State Children Cohort study. Dual-energy x-ray absorptiometry (DXA) was used to assess abdominal obesity, as measured by android/gynoid fat ratio (A/G ratio), android/whole body fat proportion (A/W proportion), visceral (VAT) and subcutaneous fat (SAT) areas. Continuous metabolic syndrome score (cMetS), calculated as the sum of the age and sex-adjusted standardized residual (Z-score) of five established MetS components, was used to assess the MetS burden. Linear regression models were used to analyze the impact of DXA measures on cMetS and individual cMetS components. All models were adjusted for age, race, sex, and general obesity.

RESULTS—Abdominal obesity is significantly associated with increased cMetS. With 1 standard deviation (SD) increase in A/G ratio, A/W proportion, VAT area, and SAT area, cMetS increased by 1.34 (SE=0.17), 1.25 (SE=0.19), 1.67 (SE=0.17), and 1.84 (SE=0.20) units, respectively. At individual component level, strongest association was observed between abdominal obesity and insulin resistance than lipid-based or blood pressure-based components. VAT and SAT had a stronger impact on insulin resistance than android ratio-based DXA measurements.

CONCLUSIONS—Abdominal obesity is associated with higher MetS burden in adolescent population. The association between abdominal obesity and insulin resistance measure is the

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strongest, suggesting the key impact of abdominal obesity on insulin resistance in adolescents Mets burden.

Keywords

Abdominal Obesity; Metabolic Syndrome Burden; Insulin Resistance

INTRODUCTION

Childhood and adolescents obesity is an epidemic in United States, with a prevalence of 16.9% in 2009–2010 (1). Obesity is associated with increased risk of type 2 diabetes (T2D) and cardiovascular disease (CVD) (2–4). Recently, it has also been recognized that excessive fat accumulation in abdominal region is associated with cardiometabolic risk (5). However, most of the previous studies used indirect measurements, including skinfold thickness (6), underwater weighing (7), waist circumference (WC) (8), waist-to-hip ratio (WHR) (9), and waist-to-height ratio (WHtR) (9), to estimate the abdominal fat distribution. Comparing to the anthropometric measurements, dual-energy x-ray absorptiometry (DXA) provides direct and accurate measurements of the abdominal fat distribution. This method has been validated against various “gold standard”, including underwater weighing (10), computed tomography (11), and magnetic resonance imaging (12). Because of the low radiation and cost, DXA is a practical tool to quantify the abdominal fat distribution.

Metabolic syndrome (MetS), a clustering of multiple cardiometabolic abnormalities, is associated with increased T2D and CVD risk (13,14). Data from the U.S. National Health and Nutrition Examination Survey (NHANES) indicates the prevalence of MetS in adolescents increased from 4% to 9% from 1988 to 2006 (15,16) and the prevalence among obese children and adolescents were approximately 30–50% (17,18). However, the results from these studies were not comparable, due to lack of a universal definition of MetS in children and adolescents. In addition, binary definitions of MetS in children and adolescents are subject to the loss of information contained in the individual components, which are all continuous variables. To overcome these limitations, several methods (19–21) have been used to derive a continuous metabolic syndrome score (cMetS) and represent an aggregated burden for MetS in healthy adolescents.

To date, the association between abdominal obesity and MetS burden in healthy adolescents is unclear. We conducted this study to investigate the association between DXA-measured abdominal adiposity and MetS burden, assessed by cMetS, in a population-based sample of healthy adolescents.

METHODS

Population

We used available data from 421 adolescents who completed the follow-up examination of Penn State Children Cohort (PSCC) study. The baseline recruitment and examine procedures have been published elsewhere (22). A total of 700 children aged 5–12 years participated in baseline examination, conducted in 2002–2006. Among the 700 subjects, 421

completed the follow-up examination during 2010–2013, yield a response rate of 60%. The loss to follow-up was mainly due to subjects moving out of the central Pennsylvania area. However, there was no major difference in the baseline demographic characteristics between those who participated in the follow-up study and those who did not. The study participants were examined in the Clinical Research Center in the Pennsylvania State University College of Medicine. After undergoing a whole-body DXA scan, a detailed physical examination and questionnaire-based data collection protocol were performed. The participants stayed overnight in a sleep laboratory to complete a standardized polysomnography recording. Morning blood, saliva, and urine samples were collected after the overnight fasting. The study protocol was approved by Penn State University College of Medicine Institutional Review Board. Written informed consents were obtained from participants and their parents or legal guardians if younger than 18 years.

Dual-Energy X-Ray Absorptiometry

Whole-body DXA scan was performed using a Hologic Discovery W scanner (Hologic Inc., Waltham, MA). DXA scan uses two beams of low-energy x-rays that are collected by the detectors after attenuation by the body tissue through which they have passed. Subjects were required to remove all metal, plastic, and rubber materials to avoid effect on x-ray beams. Android region, gynoid region, visceral adipose tissue (VAT), and subcutaneous adipose tissue (SAT) were selected as regions of interest (ROI). Briefly, the height of the android ROI was defined as 20% of distance between the iliac crest and subject's chin. The height of gynoid ROI is 2 times the height of the android ROI. The upper boundary is 1.5 times height of android ROI below the iliac crest. The width of each ROI was adjusted to include all tissue in the region. VAT and SAT were measured in an area overlaps android region. DXA directly measures SAT on each side of the abdominal cavity. By using appropriate modeling, the amount of SAT over the visceral cavity can be estimated and subtracted from the total abdominal fat to get VAT. All ROI were identified by Hologic APEX 4.0 software (Hologic Inc., Bedford, MA) and verified by an experienced investigator. Android/gynoid fat mass ratio (A/G ratio), android/whole body fat mass proportion (A/W proportion), gynoid/whole body fat mass proportion (G/W proportion), VAT, and SAT areas were used in this report.

Continuous Metabolic Syndrome Score

cMetS, derived from a Z-score approach, was used to represent the MetS risk. To be consistent with the adults MetS criteria, 5 established MetS components—WC, mean arterial pressure (MAP), homeostasis model assessment of insulin resistance (HOMA-IR), triglycerides (TG), and high-density lipoprotein (HDL)—were included to quantify metabolic burden. WC and three seated blood pressure readings were measured during physical exam. The average of the 2nd and 3rd blood pressure measurements was used. MAP was calculated as diastolic pressure+1/3 systolic pressure. Glucose, insulin, and lipids profiles were obtained from fasting blood sample. HOMA-IR was calculated as (fasting insulin level*glucose level)/22.5. Because HDL level is inversely related to metabolic risk, it was multiplied by -1. The five individual components were age and gender-adjusted. The five adjusted components were converted to Z-scores then summed to create the cMetS. A higher score indicates a higher MetS burden.

Other Covariates

Subjects' demographic information was collected via self-administered questionnaires. Subjects' height and weight were measured to calculate body mass index (BMI) percentile. BMI percentile was adjusted for age and gender based on the formula from the 2000 U.S. CDC growth charts.

Statistical Analysis

Among 421 subjects, 47 were excluded from analysis due to incomplete data, yield an effective sample size of 374. T-tests and chi-square tests were used to compare the gender-specific distribution of demographic characteristics. Pearson correlation coefficient was used to represent the correlation among DXA measurements and BMI percentile. Linear regression models were used to evaluate the association between DXA and cMetS. A/W proportion and G/W proportion entered the same model, allowing them to control for each other. To evaluate the effect of abdominal adiposity on the individual MetS risk factors, linear regressions were performed on each of the cMetS components. Logarithmic-transformation was performed on HOMA-IR to achieve a normal distribution. To compare the magnitude of the impact of DXA measurements on each cMetS component, both DXA and cMetS component variables were standardized to their standard deviation (SD). Therefore, the regression coefficients (β) from these standardized regression models were expressed as the impact of 1 SD increase in DXA variable on the shift in distribution of cMetS components. We adjusted for BMI percentile as a measure of general obesity in all final models to focus on the associations of central obesity and MetS burden. SAS statistical package (Version 9.3; SAS Institute, Cary, NC) was used for all analyses.

RESULTS

Table 1 presents the summary statistics of the demographic characteristics of the study population. The mean (SD) age of the population was 16.87 (2.19) years, with 77.54% whites and 53.74% male. The mean follow-up time was 7.70 years. Males had a higher proportion of android fat but lower gynoid fat. Females showed a significantly higher SAT area than males. The average cMetS score was 0.08 (3.03). No significant difference was found in cMetS score between males and females.

Pearson correlation coefficients among DXA measurements and BMI percentile (Table 2) suggested that VAT area highly correlated with A/G fat ratio and A/W fat proportion (0.94 and 0.84, respectively). Whereas, there were only moderate correlation between DXA measures and BMI percentile. Table 3 summarized the results from linear regression models for the association between 1 SD increase in DXA measurements and cMetS. The android-related measurements, VAT, and SAT showed significant positive relationship to cMetS. G/W proportion showed a negative but not significant relationship to cMetS. VAT and SAT showed a stronger impact on cMetS than A/G and A/W ratios. The associations remained similar after adjusting major confounders, including BMI percentile. Standardized regression coefficients relating 1 SD increase in DXA measurements and the shift in the distribution of cMetS components are presented in Table 4. Android, visceral, and subcutaneous obesity were associated with increased WC, TG, HOMA-IR, and lower HDL

(all $p < 0.01$). When comparing the β s of each DXA measurement on HOMA-IR, VAT area ($\beta = 0.42$) and SAT area ($\beta = 0.53$) showed a stronger impact than A/G fat ratio ($\beta = 0.31$) and A/W fat proportion ($\beta = 0.24$). When comparing the β s for the same DXA variable on different cMetS components, our results suggested that abdominal obesity had a stronger impact on HOMA-IR (e.g. SAT area: $\beta = 0.53$) than TG ($\beta = 0.37$) and HDL ($\beta = -0.33$). The association between DXA measures and BP was less consistent and less pronounced.

DISCUSSION

Recent studies have shown associations between childhood MetS and increased risk of T2D and CVD in adulthood (13,14). Therefore, controlling the MetS burden in children may prevent onset of T2D and CVD in later life. However, the lack of universal definition of MetS in adolescents complicates the comparisons and interpretations of different studies, which contributes to our understanding of the public health burden of MetS. For example, Cook et al. and de Ferranti et al, respectively, reported a 4.2% and 9.2% MetS prevalence in the U.S. adolescents using the same NHANES III data but different MetS criteria (15,23). To avoid the dilemma, and to overcome the loss of information when reclassifying continuous variables into binary variables, we used the cMetS score to assess the MetS burden in our sample of adolescents. In this study, we found adiposity, especially abdominal obesity, is a risk factor of elevated cMetS, independent from general obesity. This finding suggests abdominal adiposity is significantly associated with increased MetS burden, even in healthy adolescents. To the best of our knowledge, this is the first study associating abdominal obesity and MetS burden in healthy adolescents.

At individual component level, abdominal adiposity has been associated with dyslipidemia, hypertension, and insulin resistance (IR) in children (24–28). Daniels et al. studied a sample of 127 children and adolescents and found an association between abdominal adiposity, measured by fat mass, and TG, HDL, but not LDL (24). Recently, a study conducted in 164 Japanese school children found WHtR-measured abdominal obesity is associated with decreased HDL, elevated TG and HOMA-IR (25). Data from a sample of 66 obese children and adolescents showed DXA-measured A/G ratio is associated with IR (26). In another study performed in 61 prepubertal children, investigators found SAT and VAT were associated with higher TG and insulin level (27). Lurbe et al. reported a positive relationship between WHR and BP in a sample of 140 children aged 6–16 (28). We obtained similar findings from this study. More importantly, the standardization of both DXA and cMetS components to their respective SDs allowed us to compare the strength of the associations, not only the impact of different abdominal adiposity measurements on the same cMetS component, but also the effect of one central obesity measurement, e.g. VAT area, across five cMetS components. As expected, the strongest associations were found between central obesity and WC, as both DXA and WC are physical measurements of the dimension of waist area. When comparing the impact of different DXA variables, VAT and SAT showed a stronger impact on IR, than the traditional android fat based measures. However, android, visceral, and subcutaneous fat have similar impact on lipid-based components of cMetS. Most importantly, when comparing the effect of central obesity on different cMetS components, the strongest metabolic impact of central obesity was IR, followed by lipids measures, and the impact on blood pressure was less consistent and less pronounced. The

less consistent and weaker association may due to the young age of this healthy population. We anticipate a consistent association between central obesity and elevated blood pressure in the adulthood, if the central obesity persists. Since we adjusted for BMI percentile as a measure of general obesity in all of our final models, the abdominal obesity and MetS burden associations are reflective of central obesity, independent of the general obesity.

To date, the key impact by which abdominal fat accumulation affects MetS risk in children and adolescents is unclear. It has been postulated that visceral fat induced IR plays a critical role (5,29,30). Visceral obesity has been related to excess release of non-esterified fatty acids. On the other hand, visceral fat is more lipolytic than subcutaneous fat and is also less sensitive to anti-lipolytic effect of insulin. These unique characteristics of visceral adipocyte, combined with proximity of the liver to the visceral fat depot, probably result in an increased delivery of fatty acids to the liver. The excess fatty acids buildup in the liver leads to a higher triglycerides production (5). As one of the risk factors for low HDL, IR has been associated with an increased catabolic rate of HDL and impaired maturation of HDL particles (29). Several links between IR and elevation blood pressure have been suggested. Reaven suggested that insulin-mediated sympathetic stimulation may represent a compensatory mechanism aimed at limiting weight gain by increasing thermogenesis (30), the effects of hyperinsulinemia and increased sympathetic activity on the kidney and cardiovascular system tend to increase the blood pressure. Other mechanisms involve the effect of insulin on the handling of sodium and water by the kidney and endothelium-dependent vasodilation (31). Currently, most literature suggest that abdominal, particularly visceral, obesity leads to IR, and through IR, lead to a pro-atherogenic lipids profile, elevated blood pressure and fasting glucose. As a result, central obesity increases the risk of CVD and T2D. In our study, since both WC and DXA measure the abdominal physical dimension, it is anticipated that WC and DXA are strongly associated. However, the strong and consistent associations between DXA measures and HOMA, TG, and HDL-cholesterol suggest the key impact of central obesity on IR in MetS burden. To investigate the potential mediating effect of IR in the relationship between visceral fat and other metabolic syndrome components, we compared the regression coefficients from VAT on MAP, TG and HDL as presented in Table 4 and those from models also adjusted for insulin resistance. The adjustment of insulin resistance attenuated the magnitude of associations from VAT area by 25%, 23%, and 11%, respectively for MAP, TG, and HDL-cholesterol. However, the regression coefficients of VAT in these insulin resistance-adjusted models remained statistically significant (data not shown). The substantial attenuations of the VAT impact on other metabolic syndrome components after adjustment of insulin resistance indicate the mediating role of insulin resistance in the central obesity and metabolic syndrome relationship, at least partially.

With longer follow-up data, we anticipate that such association will become more consistent. We also anticipate that the central obesity in adolescents, if left unmanaged, will lead to increased T2D and CVD risk in adulthood.

When comparing to previous studies, one of the strengths of this study is the use of DXA to directly measure adipose tissue distribution. For example, WC, WHR, and WHtR have been used in previous studies to estimate fat distribution and the association with various cardiometabolic morbidities (8,9). In other studies, such as the Bogalusa Heart Study (6),

skinfold thickness was used as a surrogate of body composition. Their studies relied on indirect anthropometric measurements, which may not be representative of true body composition and distribution of adipose tissue. More importantly, compared to previous studies used DXA measure, which only provided region-specific fat mass, the latest technique enabled us to investigate the distinction between visceral and subcutaneous abdominal tissues. This more accurate and precise measurement allows a better assessment of the effect of fat deposition. While the DXA imaging technology has been approved for clinical use to assess bone density, it has not been approved for clinical use to assess central obesity. However, using DXA to measure the abdominal fat distribution has been widely used for research purpose. Once DXA is approved for clinical use to assess abdominal obesity, the significant association between abdominal obesity and cMetS presented in these population-based data may be useful in design specific interventions to target abdominal obesity. This argument is also supported by the moderate correlation between abdominal obesity and the general obesity as measured by BMI percentile as shown in Table 2. It is obvious that the binary Mets diagnosis criteria developed for adult are not appropriate for healthy adolescent population. We, therefore, choose to use cMetS score to represent the continuous MetS burden in adolescents, using all five components that are used in the adult MetS definition. The consistency enables the possibility of tracking the MetS risk across the lifespan. Meanwhile, the Z-score accounted for the influences of age and sex on each individual risk factor and the MetS burden.

It should be noted there are some limitations to the current study. First, the response rate of current follow-up study is 60%. The results may be biased by the relatively low response rate. Second, the five cMetS components were equally weighted and treated independently when summing up the individual Z-score. It may not reflect the true association between individual risk factors and MetS risk, since IR may play the most critical role in the association. Third, the cMetS derived in this study cannot be compared to other study, since it is sample specific. However, we may argue that the present population-based sample of adolescents represents the demographic characteristics of central Pennsylvania. Therefore, the result is generalizable to adolescent population and comparable to other studies targeting the same population.

To sum up, in this population-based sample of adolescents, we found that abdominal obesity, independent from general obesity, is associated with a significantly higher MetS burden. If central obesity persists, it may lead to clinical MetS in adulthood. At individual component level, this study confirms that excess abdominal adipose tissue is an independent risk factor for higher burden of insulin resistance as reflected by HOMA, triglycerides, and lower HDL. If the insulin resistance persists, it may lead to T2D diabetes and CVD in later life. Future studies with long-term follow-up are needed to investigate the pathophysiological link between abdominal obesity and unfavorable cardiometabolic alternations and T2D. Most importantly, longer term follow-up studies may shed light on the importance of reducing central obesity in adolescents on the prevention of T2D and CVD in adulthood.

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Table 1

Demographic status of the study population

	All (N=374)	Male (N=201)	Female (N=173)	P value
Age (years)	16.87 (2.19)	16.76 (2.17)	16.99 (2.20)	0.30
Years of follow up	7.70 (1.37)	7.75 (1.36)	7.64 (1.39)	0.41
White (%)	77.54	75.72	79.10	0.43
BMI percentile	65.71 (28.27)	64.69 (29.67)	67.16 (26.71)	0.40
A/G fat ratio	0.36 (0.11)	0.38 (0.11)	0.33 (0.10)	<0.01
A/W fat proportion	0.06 (0.02)	0.06 (0.02)	0.06 (0.01)	0.74
G/W fat proportion	0.18 (0.02)	0.16 (0.02)	0.19 (0.02)	<0.01
VAT area (cm ²)	59.93 (39.48)	62.48 (36.70)	57.62 (42.88)	0.24
SAT area (cm ²)	215.96 (157.03)	156.87 (138.95)	287.55 (149.53)	<0.01
cMetS	0.08 (3.03)	0.15 (2.76)	0.08 (3.36)	0.82
WC (cm)	80.38 (13.56)	82.72 (14.10)	77.88 (12.71)	<0.01
MAP (mm Hg)	82.37 (9.14)	82.98 (9.01)	82.00 (9.27)	0.30
TG (mg/dL)	95.04 (47.98)	95.70 (51.09)	95.40 (44.96)	0.95
HDL (mg/dL)	49.62 (12.56)	45.94 (10.16)	54.04 (13.81)	<0.01
Log-HOMA	-0.22 (0.78)	-0.27 (0.69)	-0.16 (0.88)	0.18

Results were expressed as mean (SD) and percentage for continuous and categorical variables, respectively.

Table 2

Pearson correlation coefficients among BMI percentile and DXA measurements

	BMI percentile	A/G fat ratio	A/W fat proportion	G/W fat proportion	VAT area	SAT Area
BMI percentile	1.00					
A/G fat ratio	0.54	1.00				
A/W fat proportion	0.61	0.91	1.00			
G/W fat proportion	-0.12	-0.63	-0.26	1.00		
VAT area	0.64	0.94	0.84	-0.37	1.00	
SAT Area	0.65	0.67	0.77	-0.09	0.81	1.00

Table 3

Regression coefficient (SE) and p value in association between 1 SD increase in DXA

DXA variable	Model 1		Model 2	
	β (SE)	P value	β (SE)	P value
A/G fat ratio	1.67 (0.14)	<0.01	1.34 (0.17)	<0.01
A/W fat proportion	1.60 (0.14)	<0.01	1.25 (0.19)	<0.01
G/W fat proportion	-0.18 (0.15)	0.21	-0.20 (0.17)	0.23
VAT area	1.83 (0.13)	<0.01	1.67 (0.17)	<0.01
SAT area	1.67 (0.13)	<0.01	1.84 (0.20)	<0.01

Model 1: Unadjusted model

Model 2: Adjusted for age, race, gender, and BMI percentile.

Table 4

Regression coefficient (SE)[†] and p value in association between 1 SD increase in DXA measurements and corresponding shift in the distribution of individual cMetS components

	WC SD=13.56 cm	MAP SD=9.14 mmHg	Log-HOMA SD=0.78	TG SD=47.98 mg/dL	HDL SD=12.56 mg/dL
A/G fat ratio	0.49 (0.03)	0.11 (0.06)	0.31 (0.06)	0.38 (0.06)	-0.27 (0.06)
A/W fat proportion	0.44 (0.04)	0.08 (0.07)	0.24 (0.07)	0.36 (0.07)	-0.25 (0.07)
G/W fat proportion	-0.13 (0.03)	-0.05 (0.06)	-0.08 (0.06)	-0.04 (0.06)	0.08 (0.06)
Visceral fat area	0.70 (0.03)	0.16 (0.06)	0.42 (0.06)	0.39 (0.07)	-0.28 (0.07)
Subcutaneous fat area	0.79 (0.04)	0.15 (0.07)	0.53 (0.07)	0.37 (0.08)	-0.33 (0.08)

Adjusted for age, race, sex, and BMI percentile.

[†]: The regression coefficient (SE) was expressed as the effect of 1 SD increase in DXA variables on the shift in distribution of cMetS variables. A regression coefficient (SE) =0.49 (0.03) suggested with 1 SD increase A/G fat ratio, the WC would increase by 0.49 (0.03) of its SD.

* : p<0.05;

** : p<0.01