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Anthropometric measures of abdominal adiposity for the identification of cardiometabolic risk factors in adolescents

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Summary

Sagittal abdominal diameter (SAD) was obtained in 65 adolescents referred for assessment of cardiometabolic risk. We found that SAD was associated with cardiometabolic risk factors independent of BMI in males, but that SAD was not superior to BMI or other measures of abdominal adiposity for the detection of metabolic syndrome.

Introduction

BMI is widely used to identify individuals with excess adiposity who may be at risk for cardiometabolic disease [1]. However, the use of BMI to screen for excess adiposity in children is limited by changes in body composition with growth and development [2]. Moreover, BMI does not provide information about abdominal fat. Abdominal obesity reflects excess subcutaneous and visceral adipose tissue (VAT). VAT is thought to be more metabolically active and predictive of cardiometabolic risk [3]. Measures of abdominal obesity including waist circumference (WC), sagittal abdominal diameter (SAD) and waist/hip ratio (WHR) have been shown to be strongly correlated with VAT [4, 5] and

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cardiometabolic risk factors [6]. In adults, SAD may be a more accurate measure of VAT [5] and better predictor of cardiometabolic disease [7, 8] than other anthropometric measures. The relationship between SAD and cardiometabolic risk in children remains unclear. Our objective was to evaluate the ability of SAD compared to WC, WHR, and BMI to identify the presence of cardiometabolic risk factors in a high-risk population of youth.

Methods

This study was approved by the IRB of The Children's Hospital of Philadelphia. 65 participants (26 male) aged 11-17 years referred to endocrinology for evaluation of cardiometabolic risk were recruited. Weight, height, SAD, WC, and WHR were obtained as previously described [5, 9]. BMI Z-scores were calculated [10] and categorized according to current recommendations [11]. Blood samples for cardiometabolic risk factors were obtained in the fasting state (**Supplemental Methods**). HOMA-IR was calculated as $\text{HOMA-IR} = [\text{fasting plasma insulin } (\mu\text{IU/mL}) \times \text{fasting plasma glucose (mmol/L)}] / 22.5$. Metabolic syndrome (MetSyn) was defined using the International Diabetes Federation Criteria [12].

Means/medians were compared by two-sample t-tests or Wilcoxon signed-rank tests; proportions by χ^2 analysis. Outcomes were normalized by log transformations where indicated. Pearson and partial correlation coefficients were determined associations between measures of abdominal adiposity and cardiometabolic outcomes; tests of Hotelling's T and Steiger's Z statistics were used to compare strength of correlations. Receiver operator characteristic (ROC) curves were used to calculate the area under the curve (AUC) for anthropometric measures to identify MetSyn; χ^2 tests were used to compare AUC values. Analyses were performed using Stata 12 (StataCorp, LP, College Station, TX).

Results

Participant characteristics are shown in **Supplemental Table 1**. Blacks comprised 68% of the sample. Mean BMI-Z was 2.2 ± 0.3 ; 63/65 participants were obese, 2/65 were overweight. 14% of participants had MetSyn [12].

Significant correlations ($p < 0.05$) were seen between all measures of abdominal adiposity and HOMA-IR, insulin, CPEP, and triglycerides in males, and hemoglobin A1C (HbA1c) in females (**Table 1**). Additionally, in females significant correlations were seen between WHR and CPEP and triglycerides and WC and HDL. In males, after adjusting for BMI-Z, significant correlations remained between SAD, WC, WHR and HOMA-IR, insulin, and CPEP. In females, after adjustment for BMI-Z, only the correlation between triglyceride level and WHR remained significant. The correlation between SAD and CPEP (0.775) in males was stronger than the correlation between BMI or BMI-Z and CPEP (0.612, 0.521).

The ability of SAD to identify MetSyn using AUC values (**Table 2**) in males (0.605) and females (0.648) did not differ from other measures of abdominal adiposity or BMI. The addition of SAD to BMI or BMI-Z did not result in a greater AUC compared to BMI or BMI-Z alone. In females, the AUC for WC (0.778) and BMI-Z with WC (0.833) was greater than the AUC for BMI-Z alone (0.657).

Conclusions

SAD in males was significantly correlated with several cardiometabolic risk factors independent of BMI, but the strength of those correlations did not differ from other measures of abdominal adiposity. Notably, CPEP had a stronger correlation with SAD compared to BMI in males. CPEP may be an early marker of impaired glucose metabolism in obese adolescents [13] raising the possibility that SAD could improve upon BMI for the early detection of diabetes risk. After adjustment for BMI, SAD was not significantly correlated with any cardiometabolic risk factors in females.

ROC curve analysis revealed that SAD alone or in conjunction with BMI did not improve identification of MetSyn compared to BMI or other measures of abdominal adiposity. The use of WC (either alone or with BMI) was superior to BMI for the detection of MetSyn in females, suggesting a role for the assessment of abdominal adiposity in this population. Findings of previous studies comparing the ability of BMI and measures of abdominal adiposity to identify cardiometabolic risk factors are conflicting. WC has been found to be associated with cardiometabolic risk factors independent of BMI in some studies [14, 15] while others have found that measures of abdominal adiposity are not better than BMI for the identification of cardiometabolic risk [16, 17]. A recent study reported measures of abdominal adiposity were not better predictors of VAT than BMI in children [18], which may explain these negative findings.

The results of our study may be limited by the high prevalence of obesity in our population (mean BMI-Z of 2.2). It is possible that measures of abdominal adiposity may be more useful predictors of cardiometabolic risk in non-obese individuals where excess abdominal adiposity could be missed if using a whole body measure such as BMI [19]. Future studies of children with a range of BMI are needed to identify the populations in which measurements of abdominal adiposity including SAD will improve cardiometabolic risk assessment. SAD has been added to the National Health and Nutrition Examination Survey procedures [20] which will provide opportunities for such studies. In summary, our results suggest that in an obese pediatric population, SAD is correlated with a number of cardiometabolic risk factors, but it does not improve upon BMI, WC, or WHR for the identification of MetSyn.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

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Table 1

Correlations and partial correlations between anthropometric measures of adiposity and cardiometabolic outcomes

Males											
Pearson Correlation Coefficients				Partial Correlation Coefficients (adjusted for BMI)				Partial Correlation Coefficients (adjusted for BMI-Z)			
BMI	BMI-Z	SAD	WC	WHR	SAD	WC	WHR	SAD	WC	WHR	WHR
Homa IR ^I	0.452 ²	0.356	0.559 ³	0.530 ⁴	0.530 ³	0.316	0.353	0.483 ²	0.458 ²	0.425 ²	
Insulin ^I	0.669 ⁴	0.610 ³	0.762 ⁴	0.761 ⁴	0.630 ³	0.491 ²	0.360	0.578 ³	0.576 ³	0.396 ²	
Glucose	-0.308	-0.373	-0.294	-0.207	-0.081						
CPEP	0.612 ²	0.521 ²	0.775 ^{3,5}	0.707 ³	0.698 ³	0.453 ²	0.500 ²	0.710 ³	0.576 ³	0.551 ³	
A1c	0.141	0.193	0.053	0.065	0.207						
TRI ^I	0.602 ³	0.532 ³	0.613 ³	0.594 ³	0.579 ³	0.150	0.321	0.385	0.321	0.368	
LDL	-0.174	-0.195	-0.115	-0.204	-0.308						
HDL ^I	-0.208	-0.251	-0.119	-0.050	-0.048						

Females											
Pearson Correlation Coefficients				Partial Correlation Coefficients (adjusted for BMI)				Partial Correlation Coefficients (adjusted for BMI-Z)			
BMI	BMI-Z	SAD	WC	WHR	SAD	WC	WHR	SAD	WC	WHR	WHR
Homa IR ^I	0.024	0.142	0.059	0.152	0.245						
Insulin ^I	0.102	0.216	0.101	0.205	0.250						
Glucose	-0.026	0.120	0.005	0.046	0.172						
CPEP	0.203	0.281	0.270	0.280	0.334 ²	0.212	0.276	0.066	0.100	0.233	
A1c	0.327 ²	0.365 ²	0.413 ³	0.350 ²	0.334 ²	0.141	0.221	0.213	0.110	0.190	
TRI ^I	0.095	0.208	0.177	0.307	0.419 ²	0.448 ³	0.423 ³	0.013	0.237	0.373 ²	
LDL	0.150	0.191	0.130	0.191	0.171						
HDL ^I	-0.390 ²	-0.408 ²	-0.238	-0.404 ²	-0.253	-0.145	-0.100	0.093	-0.148	-0.067	

^I HOMA-IR, insulin, triglycerides, and HDL were normalized using logarithmic transformation. Spearman correlation coefficients were also performed on non-transformed variables and they did not differ substantially from values reported above.

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- ² Statistically significant correlation, $p<0.05$
- ³ Statistically significant correlation, $p<0.01$
- ⁴ Statistically significant correlation, $p<0.0001$
- ⁵ Statistically stronger correlation compared to BMI and BMI-Z, assessed by Hotelling's T and Steiger's Z, $p<0.05$

Table 2

AUC for measures of abdominal adiposity and BMI to identify metabolic syndrome in males and females

	AUC			AUC	
	Males	Females		Males	Females
BMI-Z	0.456	0.657	BMI	0.590	0.593
SAD	0.605	0.648	SAD	0.605	0.648
WC	0.561	0.778 ¹	WC	0.561	0.778
WHR	0.614	0.833	WHR	0.614	0.833
BMI-Z+SAD	0.728	0.639	BMI+SAD	0.772	0.639
BMI-Z+WC	0.623	0.833 ²	BMI+WC	0.614	0.889
BMI-Z+WHR	0.597	0.852	BMI+WHR	0.614	0.843

¹ Statistically significant difference from BMI-Z, p=0.03² Stastically significant difference from BMI-Z, p=0.02