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Visceral Adiposity Index and Lipid Accumulation Product Related to Insulin Resistance and Metabolic Syndrome in Obese College Students

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Abstract

BACKGROUND: Visceral obesity in adolescent girls increased the risk of cardiometabolic disease. A simple indicator has been developed to assess metabolic risk through visceral adiposity index (VAI) and lipid accumulation product (LAP) in people with visceral obesity.

AIM: This study analyzed the relationship between VAI and LAP with insulin resistance (IR) and metabolic syndrome (MS) in obese female adolescents.

METHODS: An observational study was conducted on 120 students at Universitas Diponegoro. VAI was obtained from calculation formulations which includes body mass index, waist circumference (WC), triglycerides (TGs), and High-density lipoprotein (HDL) cholesterol. LAP was obtained from calculation formulation which include WC and TGs. IR values were obtained based on the calculation of homeostasis for assessment models IR. The metabolic syndrome score (cMetS) was obtained by the calculation of components of WC, blood pressure, fasting blood glucose, TG, and HDL cholesterol. Bivariate analysis using the Spearman Rank correlation test.

RESULTS: We found that 40% of subjects had a VAI score that was considered at risk. As many as 20.8% of obese adolescent girls experience MetS while the other 79.2% are pre-metabolic syndrome and 83.3% of subjects have experienced IR. VAI showed a significant relationship with IR ($p \leq 0.001$; $r = 0.667$) and cMetS ($p = 0.007$; $p = 0.245$). LAP showed a significant relationship with IR ($p < 0,001$; $r = 0,385$) and MS ($p < 0,001$; $r = 0.372$).

CONCLUSION: We found that VAI and LAP could be an indicator for estimating IR and MS in obese female adolescent.

Introduction

Obesity is a chronic disease caused by multiple complex factors [1]. Obesity is defined as the excessive accumulation of body fat due to an increase in adipose tissue which increases body weight [2]. Obesity can occur in any age group including adolescents [3]. Causes of obesity in adolescents can be due to low physical activity, sedentary lifestyle, high-calorie, and fat diets, resulting in an imbalance between the amount of energy gained with the energy expended resulting in increased accumulation of fat in body tissues [4], [5]. Obesity is the initial trigger for more serious health problems such as metabolic syndrome, diabetes mellitus (DM), hypertension, coronary heart disease, depression, osteoarthritis, cancer, and even death [6], [7].

Data from National Health and Nutrition Examination Survey (NHANES) states that 33.8% of adults (aged 20 years or older) and 16.8% of children and adolescents (aged 2–19 years) are obese [8]. This is supported by research in Iran in 2010 which said that the total prevalence of central obesity was 32.01% and significantly higher in women (57.2%) compared to men (15.8%) [9]. Research in Semarang on 516 teenagers in Public Senior High School 15 Semarang shows that 66 people (12.8%) are obese adolescents, 46 people (8.9%) are adolescents with central obesity, and there are 19 adolescents (47.5%) with metabolic syndrome from obese adolescents [10].

Obesity especially android type or central obesity is a type of obesity that is the most dangerous risk compared to other types [11]. Central obesity is associated with increased adipocytokine production, proinflammatory activity, decreased insulin sensitivity, increased risk of diabetes, dyslipidemia (high-levels of triglycerides [TGs] and low-levels High-density lipoprotein [HDL]), hypertension, atherosclerosis, and higher mortality rates) [10]. People with central obesity have fat cells deposited around the stomach releasing fat into blood vessels thereby increasing the risk of developing metabolic syndrome and cardiovascular disease [12]. Many of studies have shown that increased fat in the stomach area is directly related to the increase in systemic inflammation with the production of various inflammatory cytokines by adipocytes [13].

In the past few years, new parameters have been developed to estimate the existence of obesity and the distribution pattern of adipose tissue, especially visceral ones; such as lipid accumulation products (LAP) proposed by Kahn in 2005 [14], which is a mathematical model which studied the connection between waist circumference (WC) as the anthropometric variable with fasting TG levels. Furthermore, the visceral adiposity index (VAI), discovered by Amato in 2010 [10], made it possible to distinguish the degree of adipose tissue dysfunction, using WC, fasting TG levels, and Body Mass Index (BMI), and HDL cholesterol levels.

Central obesity is associated with visceral fat accumulation which is correlated positively with total cholesterol, LDL, TGs, hyperinsulinemia, and inversely related to insulin sensitivity and HDL levels during childhood and adolescence. VAI and LAP are strongly associated with adipocytokine synthesis, proinflammatory activity, insulin resistance (IR), dyslipidemia, hypertension, and atherosclerosis [13]. VAI and LAP are useful indicators to understand and depicture adiposity not only about the relationship between body weight and height but also its location and function. Too much accumulation of visceral fat could be related to many comorbidities associated with obesity [13].

Metabolic syndrome which can cause cardiovascular disease is often associated with visceral obesity and IR [15]. Visceral adipose tissue produces various bioactive substances, namely, adipokines that affect metabolism and trigger systemic inflammation by active endocrine function and lead to IR. This IR then leads to the development of atherogenic dyslipidemia profiles and prothrombotic and proinflammatory conditions [6]. Dyslipidemia in individuals with IR is characterized by an increase in TGs, apolipoprotein B, low-density lipoprotein (LDL) particles, and a decrease in HDL concentrations which become smaller HDL particles [6], [16]. The results of inflammation and IR in obese adolescents are metabolic complications that increase death's risk factors due to increased risk of Type 2 DM, hypertension, dyslipidemia, and atherosclerosis [5], [17]. Hence, effective indicators are necessary to detect the distribution of visceral fat function in obese adolescents so that IR and metabolic syndrome risks can be treated early. The aim of this study is to analyze the relationship between VAI and LAP with IR and metabolic syndrome in obese adolescents.

Methods

This study was an observational study with cross-sectional design conducted from June to August 2019. The target population in this study were young women aged 17–21 years in Semarang and the accessible population were young women in the Universitas Diponegoro campus area. Based on sample calculation using the correlation test formula with 10% drop out the correction a minimum sample size of 85 subjects was obtained. The sampling method in this study was The Cluster random sampling technique. Based on the inclusion criteria, young women aged 17–21 years, willing to be the subjects of this study and filled out informed consent, had a WC of 80 cm, were not pregnant and not doing breastfeeding, did not smoke and consume alcohol, currently were not consuming any drugs which could affect cholesterol levels, we obtained 120 subjects. In the process, there was no sample that dropped out.

Screening data such as name, date of birth, address, telephone number, and smoking history were obtained through the screening form. The independent variables in this study were the visceral adiposity index (VAI) and LAP, while the dependent variables were IR (HOMA-IR) and metabolic syndrome scores (cMetS). VAI was a gender-specific mathematical index based on the calculation of parameters including BMI, WC, TG levels, and HDL cholesterol [18]. The VAI calculation formula was as follows [19]:

$$\text{Woman}: \text{VAI} = \left(\frac{\text{WC}}{39{,}68 + (1{,}88 \times \textit{BMI})}\right) x \left(\frac{\text{TG}}{1{,}03}\right) x \left(\frac{1{,}31}{\textit{HDL}}\right)$$

LAP was a simple and effective indicator that could reflect visceral obesity based on increased TGs levels and WC [20]. The value of LAP depended on the value of one's WC and TGs level, the greater the value of one's TGs and WC, the greater also the LAP value. The LAP calculation formula for female subjects was as follows [14]:

LAP = [Waist circumference (cm)–58]×Triglycerides (mmol/L)

The VAI threshold value for women <30 years old was <2.52 [21], while the normal threshold for LAP was <40.6 [22]. Body weight was measured using Omron brand digital scales with an accuracy of 0.1 kg, height was measured using microtoise with accuracy of 0.1 cm. WC was measured using a non-elastic measuring tape/metline with a maximum size of 150 cm. The WC threshold for women was ≥80 cm [23]. Blood sampling for biochemical data of HDL, TG, fasting insulin, and FBS was taken with a volume of 3 cc through the medinacubital vein after fasting for 8–10 h. Examination of HDL cholesterol levels used blood in veins. The normal HDL limit is > 40 mg/dL [24]. TGs were chemical components that could be formed from excessive fat intake in the body. TGs were in blood plasma and together with cholesterol formed plasma lipids. Tests for TG levels using a photometer with GPO method in a clinical laboratory [25]. The threshold value for TGs was <150 mg/dL [24].

IR level was obtained based on the formula which was measured using the HOMA-IR. The threshold

for the HOMA-IR value for teens was < 1,65 [26]. The HOMA-IR was calculated using the following formula [27]:

$$HOMA-IR = \frac{Fasting\ Insulin\left(\frac{mU}{L}\right) \times Fasting\ Glucose\left(\frac{mmol}{L}\right) 18}{22,5}$$

Examination of fasting insulin levels was carried out using enzyme-linked immunosorbent assay ELISA) method which was analyzed by Dynex MRX II microplate-reader at a wavelength of 450 nm [27]. Fasting blood sugar levels (FBS) examination was carried out with the polymetric enzymatic reaction method using an automatic kit that was read at a wavelength of 546 nm [27]. The threshold for fasting insulin levels was 2–25μU/L, while FBS was considered normal if it was <100 mg/dL [28].

The metabolic syndrome score (cMetS) was obtained from several components including WC, blood pressure, fasting blood glucose levels, TG levels, and HDL cholesterol levels. The cMetS measurement was carried out in several stages, consisting of: (1) Measurement of all components of cMetS, (2) standardization by changing all the metabolic syndrome components measurement results into z-score form, (3) specifically blood pressure data must be changed into mean arterial blood pressure (MAP) first through the following formula:

MAP={Systolic blood pressure–Diastolic blood pressure): 3]+Diastolic blood pressure}

(4) the standardization result of HDL cholesterol levels was inversely proportional to the metabolic risk so the HDL z-score was multiplied by −1, (5) after obtaining all z-scores from each component, then cMetS was obtained by summing all z-scores from WC, HDL levels, TGs, fasting blood glucose levels, and MAP, and (6) the results of the cMetS score ≥2.21 represent the metabolic syndrome profile [29], [30]. Blood pressure as one of the metabolic syndrome components was measured using digital Omron brand tension. Blood pressure (systole and diastole) was considered high if it was ≥130/85 mmHg [31].

Subjects characteristic data including age, weight, height, BMI, WC, TGs, HDL, VAI, FBS, insulin and HOMA-IR, systole blood pressure, diastolic blood pressure, and total syndrome score metabolic were presented in the form of minimum, maximum, median, standard deviation, and frequency distribution table. Data normality test used Kolmogorov–Smirnov. Bivariate analysis using the Spearman Rank correlation test was performed to analyze the relationship between the visceral adiposity index and LAP variables with IR. This study had been approved by the Health Research Ethics Committee Faculty of Medicine Universitas Diponegoro No.373/EC/KEPK/FK UNDIP/VIII/2019.

Research Result

Subjects characteristics

The subjects' characteristics in this study were fully described in Table 1.

Table 1: Minimum value, maximum value, median, and standard deviation

Variable	Minimum	Maximum	Median
Age (year)	17	21	19
Weight (kg)	47.8	107.4	66.6
Height (cm)	141.2	171.4	157.4
BMI (kg/m^2)	21.1	41.8	26.75
WC (cm)	80.5	114.0	85.75
TGs (mg/dL)	43.0	519.0	92.5
HDL (mg/dL)	20.0	74.0	49.0
VAI	0.6	34.9	2.25
LAP	11.2	208.2	30.8
FBS level	68.0	206.0	87.0
Insulin	2.7	52.6	10.82
HOMA-IR	0.5	18.3	2.33
Systolic blood pressure (mmHg)	87	144	110
Diastolic blood pressure (mmHg)	57	150	76
cMetS	−5.28	10.87	−0.22

cMetS: Metabolic syndrome score, LAP: Lipid accumulation product, VAI: Visceral adiposity index, BMI: Body mass index, WC: Waist circumference, TGs: Triglycerides, FBS: Fasting blood sugar, IR: Insulin resistance, HOMA-IR: Homeostasis Model Assessment of IR, HDL: High-density lipoprotein.

Table 1 showed that the subjects' age range in this study was 17–21 years. The median BMI of the subjects was 26.75 kg/m^2 and the WC of the subjects was 85.75 cm. The highest TG levels were 519 mg/dL while the lowest TG levels were 43 mg/dL. The median HDL levels were 49.0 mg/dl. The highest VAI value was 34.9 with a median value of 2.25. The highest LAP value in this study was 208.2 with a median of 30.81. The highest fasting blood glucose levels were 206 mg/dL and the highest insulin levels were 52.6μU/mL. The highest HOMA-IR value of the subjects was 18.3 with a median value of 2.33. The highest metabolic syndrome score was 10.87 and the lowest value was −5.28 with a median value of −0.22.

Based on the study results in Table 2, most of the subjects (70%) had a BMI which was classified as obese. All subjects in this study had WC in the risk category for obesity. About 86.7% of subjects had normal TG levels and only 13.3% were in the high category. HDL levels in most subjects (56.7%) were low. About 40% of research subjects were in the risk category based on the value of the visceral adiposity index (VAI). Based on LAP calculation, it was known that 29.2% of subjects had high LAP scores. FBS and insulin levels of most subjects were normal, namely, 97.5% and 89.2%, but conversely almost all subjects (83.3%) had experienced IR. There were 20.8% of subjects had metabolic syndrome while 79.2% of the subjects had metabolic pre-syndromes.

Correlation test was conducted to see the relationship between VAI with IR (HOMA-IR) and metabolic syndrome. Table 3 showed that there was significant relationship between VAI and IR ($p \leq 0.001$; $r = 0.667$) and metabolic syndrome score ($p = 0.007$; $r = 0.245$). LAP score was also found to have a significant relationship with IR (HOMA-IR) ($p < 0.001$; $r = 0.385$)

Table 2: Review of body mass index, waist circumference, triglycerides, high-density lipoprotein, visceral adiposity index, fasting blood sugar, fasting insulin, and homeostasis model assessment of insulin resistance

Characteristic	n (%)
BMI	
Normal (18.50–22.99 kg/m^2)	2 (1.7)
Overweight (23.00–27.49 kg/m^2)	34 (28.3)
Obese (> 27.50 kg/m^2)	84 (70.0)
WC	
Risk (> 80 cm)	120 (100)
TGs	
Normal	104 (86.7)
High	16 (13.3)
HDL	
Normal	52 (43.3)
Low	68 (56.7)
VAI	
Visceral obesity (≥ 2.52)	48 (40.0)
No visceral obesity (< 2.52)	72 (60.0)
LAP	
Visceral obesity (≥ 40.6)	35 (29.2)
No risk (< 40.6)	85 (70.8)
FBS level	
Normal	117 (97.5)
High	3 (2.5)
Insulin	
High	13 (10.8)
Normal	107 (89.2)
HOMA-IR	
Normal (< 1.65)	20 (16.7)
Resistant (> 1.65)	100 (83.3)
Systolic blood pressure	
Normal	117 (97.5)
High	3 (2.5)
Diastolic blood pressure	
Normal	103 (85.8)
High	17 (14.2)
MS	
Pre-MS	95 (79.2)
MS	25 (20.8)

MS: Metabolic syndrome, LAP: Lipid accumulation product, VAI: Visceral adiposity index, BMI: Body mass index, WC: Waist circumference, TGs: Triglycerides, FBS: Fasting blood sugar, IR: Insulin resistance, HOMA-IR: Homeostasis Model Assessment of IR, HDL: High-density lipoprotein.

and metabolic syndrome score ($p < 0.001$; $r = 0.720$). These results showed a positive correlation between VAI and LAP with IR and metabolic syndrome score, which meant that the higher the VAI and LAP value, the higher the IR value, and the metabolic syndrome score.

Table 3: The relationship between visceral adiposity index and lipid accumulation product with insulin resistance and metabolic syndrome

Variable	VAI		LAP	
	p	r	p	r
HOMA-IR	0.245	0.007	0.385	<0.001
cMetS	0.667	<0.001	0.720	<0.001

cMetS: Metabolic syndrome score, LAP: Lipid accumulation product, VAI: Visceral adiposity index, IR: Insulin resistance, HOMA-IR: Homeostasis Model Assessment of IR.

Discussion

The subjects of this study were 120 obese adolescent girls in an age range of 17–21 years. Based on the results, it was noted that 40% of subjects were classified as at risk of developing. The Metabolic disease with a median VAI of 2.25 [25]. VAI was a cardiometabolic indicator that used anthropometric data such as BMI and WC as well as biochemical data such as TG and HDL as its components [19], [32]. VAI, a marker of visceral adipose distribution and function, was associated with insulin sensitivity in patients with metabolic risk and showed a strong relationship with the levels of peripheral glucose utilization during euglycemic-hyperinsulinemic clamps and with visceral adipose tissue as measured by MRI [21]. VAI expressed visceral fat function consisting of changes in adipocytokine production, increased lipolysis, plasma, and free fatty acid factors [33]. VAI was an index used in clinical practice to assess cardiometabolic risk associated with visceral obesity [12]. Although VAI was not a diagnostic tool for cardiovascular and cerebrovascular events, the simplicity of measurement using parameters such as WC, BMI, TGs, and HDL made it easy to be applied [10].

Based on LAP measurement, it was known that 29.2% of subjects have a high score with a median value of 30.81 ± 22.0. LAP was simple and effective measurement index for measuring fat accumulation in the body. LAP could reflect total body fat and visceral fat function. LAP was developed as a risk of sustainability and predictors of cardiovascular disease due to excess accumulation of visceral fat in somebody with visceral obesity. The NHANES III showed that LAP had better performance than BMI in identifying high total cholesterol and LDL, low HDL, and uric acid levels in adults in the United States [14].

This study's results showed that there were 20 subjects who had a normal HOMA-IR, most subjects had normal insulin levels and normal FBS, but almost all subjects (83.3%) suffered from IR. IR was a metabolic condition when insulin becomes less sensitive which increased in the body's insulin requirements leading to hyperinsulinemia to maintain plasma glucose levels in a normal state. IR caused metabolism imbalance and was a risk factor for metabolic syndrome and other non-communicable diseases.

Bivariate analysis showed that VAI was significantly related to IR ($p = 0.007$) with a positive correlation ($r = 0.245$). These results were in line with the previous studies that specifically showed that VAI had a good predictive value for risks factor of Type 2 diabetes and hypertension which was associated with visceral adiposity. In some studies, a significant increase in VAI was associated with a significant decrease in insulin sensitivity. VAI was not an index for insulin sensitivity, but an indicator of changes in adipose function associated with IR [34]. Several recent studies also supported VAI as an indicator consisting of physical and metabolic components, namely, BMI, WC, TGs, and HDL could reflect other risk factors such as changes in adipocytokine production, increased lipolysis, and plasma free fatty acids, which could detect the distribution and function of visceral fat, IR, and increased cardiometabolic risk [5], [10].

In this study, we found a significant relationship between LAP and IR ($p < 0.001$) with a positive correlation ($r = 0.385$). These results were in line with the previous studies conducted on 1510 male subjects and 1014 female subjects. The previous study found

a significant relationship between LAP and HOMA-IR in both male subjects ($p < 0.001$; $r = 0.22$) and female subjects ($p < 0.001$, $r = 0.24$) [35]. Somebody with visceral obesity had strong correlation with IR. Visceral fat accumulation was characterized by a hyperlipolytic state with the levels of free fatty acids, which could interfere with liver function by inhibiting.

The insulin signaling process and causing IR [20], [36]. In addition, as an endocrine organ, adipose tissue could secrete adipocytokines such as TNF-α, which disrupted insulin signals and induced IR [36], [37]. LAP was a formula which was more sensitive in measuring IR than BMI and WC. Compared to LAP, BMI only represented whether somebody was overweight or not. Individuals with various degrees of Type 2 diabetes and heart disease risks could have similar BMI measurements, but different WC and metabolic profiles [20]. In addition, although WC was an index commonly used to measure obesity, WC could not distinguish between subcutaneous and visceral adipose tissue. Therefore, an increase in WC did not always reflect a high risk of accumulation of visceral fat [20]. Individuals with different levels of visceral fat might have different levels of IR risks, so indices which reflected visceral fat, such as VAI and LAP, were better in evaluating IR.

IR caused abnormalities in lipid metabolism with cholesterol levels as a risk factor for cardiovascular disease. This relationship between IR and the distribution of abdominal adipose tissue was explained in a previous study in France by Jean Vague who reported an association between android obesity and atherosclerosis and Type 2 diabetes. Following these findings, other studies had shown strong relationship between central obesity and IR, Type 2 DM and other metabolic risk factors for cardiovascular disease. The following research also showed that the relationship between increased abdominal circumference and cardiovascular risk was caused by excessive accumulation of visceral adipose tissue [12]. Decreased insulin sensitivity was not only associated with an increase in visceral fat mass but also influenced by functional factors which was indirectly reflected by TG and HDL. This condition was characterized by visceral obesity and dyslipidemia, high TGs, and low HDL which had been linked to physiological age-related leptin resistance, muscle, and liver IR, lipotoxic cardiomyopathy, and endothelial dysfunction.

This study results showed a significant relationship between VAI and metabolic syndrome ($p < 0.0001$) with a positive correlation ($r = 0.667$). In line with the previous studies in Turkey at 2012 which found that 54.5% of obese female adolescents and 45.1% of obese male adolescents with metabolic syndrome tended to have relatively high VAI scores that exceeded normal value according to their age [5]. In this study, we also found a significant relationship between LAP and metabolic syndrome ($p < 0.001$) with a positive correlation ($r = 0.720$). These results were consistent with the previous research on 683 adolescent subjects in China. In that study, it was found that there was a significant relationship between LAP and metabolic syndrome ($p < 0.001$) [37]. Other studies conducted in Spain also showed a significant relationship between LAP and the components of metabolic syndrome: HDL ($r = 0.2693$, $p < 0,0001$), WC ($r = 0.5543$, $p < 0,0001$), TGs ($r = 0.9085$, $p < 0,0001$), FBS levels ($r = 0.4036$, $p < 0.0001$), TDD ($r = 0.1226$, $p < 0.016$), but did not correlate with TDS ($r = 0.0761$, $p < 0.135$) [38].

Obesity increased IR in adipose tissue which played an important role in the pathophysiology of the metabolic syndrome [39]. Central obesity was considered as the initial cause for further disorders associated with metabolic syndrome including IR. Increased levels of free fatty acids associated with excessive adipose tissue lipolysis were important factors in the pathophysiology of IR and atherosclerosis. Increasing evidence showed that excessive activity of the hypothalamus-pituitary-adrenal axis (HPA) could play a role in the pathophysiology of abdominal obesity and IR. IR and glucose intolerance developed in 80% of Cushing's syndrome patients. Apart from Cushing's syndrome, hypercortisolism was mainly because of chronic stress. Hypercortisolism directly caused peripheral tissue IR in proportion to glucocorticoid levels. These hormonal changes caused hyperinsulinemia and increased visceral obesity resulting in dyslipidemia, hypertension, and Type 2 DM. Cortisol serum concentrations were more strongly correlated with hip ratio, TGs, total cholesterol, and leptin levels in women, compared to men with metabolic syndrome, even after some adjustments for age and BMI. Leptin had been hypothesized to have a greater influence on the women's HPA axis than in men [40].

Conclusion

Subjects with VAI classified as at risk were 40%. There were 20.8% of obese girls with metabolic syndrome while 79.2% were pre-metabolic syndrome and 83.3% subjects had IR. There was a significant relationship between VAI with IR ($p = 0.001$; $p \leq 0.001$) and metabolic syndrome in obese adolescent girls ($r = 0.27$; $r = 0.667$), LAP also showed a significant positive relationship with metabolic syndrome ($p < 0.001$; $r = 0.72$) and IR ($p < 0.001$; $r = 0.385$).

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