Relationship of Insulin Resistance with Anthropometry and Biochemical Parameters of Obese Adolescents aged 16-18

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Abstract

This cross-sectional study aimed to evaluate the relationship between anthropometry, biochemical parameters, and homeostatic model assessment (HOMA) index or insulin resistance (IR) among 97 adolescents aged 16 to 18 years with BMI > $\overline{X}$ + 2 SD kg/m^2 in Mahasarakham province, Thailand. Research protocol was approved by Mahasarakham University Ethics Committee. The participants were selected from six secondary schools in Mahasarakham province by multistage random sampling method and definite criteria and divided into two groups, obese and normal weight, based on body mass index (BMI) assessments. The participants were interviewed regarding their health history one week before completing a blood test and the investigation of nutritional aspects to check blood sugar levels, triglyceride (TG) levels, and insulin levels and possible biochemical variables were interpreted. Measurements of the proportions of the body and nutritional anthropometry were recorded. These measurements included weight, height, BMI, and biceps and triceps skinfold thicknesses. The results showed that there were differences in some variables of the obese group and the normal weight group. The obese group had statistically significant higher scores ($p< 0.05$) in weight, BMI, and volume of adipose tissue folded in biceps and triceps skinfold thicknesses with statistical significant. The researchers concluded that there was a relationship between insulin resistance and related variables in regards to the obesity of the adolescent participants. The variables of weight, BMI, and the content of adipose tissue under the biceps and triceps can induce insulin resistance received from the visceral organs. However insulin resistance has a relationship with BMI and obesity. However, the researchers did not believe that these factors clearly explained the insulin resistance condition.

Keyword: Insulin resistance, homeostatic model assessment, anthropometry and adolescents

Introduction

The prevalence of adolescent obesity in Thailand is increasing dramatically. This increase appears to be associated with an increase in the risk factors of cardiovascular disease and type 2 diabetes mellitus (T2DM), including hypertension and reduced glucose tolerance [1].

Insulin resistances (IR) as well as insulin deficiency (ID) have been shown to be strong predictors in the future development of T2DM. IR is associated with obesity, glucose intolerance or diabetes, hypertension, dyslipidemia, and cardiovascular disease. Although the relationship among these conditions is complex, IR may be the primary initiating factor [2], [3], [4]. There is increasing evidence that the onset of the IR syndrome may occur in childhood or adolescence [54], [6]. A clear definition of normal physiologic changes in IR that occur during puberty is necessary before an etiologic association between IR and other cardiovascular risk factors can be considered in this age-group.

Although obesity is an important component of IR syndrome, many people who are not obese according to standard height and weight criteria

(i.e., body mass index [BMI]) may still display features of the syndrome. Indeed, studies have indicated that normal-weight persons, whose BMI is less than 25 kg/m^2, may have as much as 40% fat in the abdominal area, a level that correlates closely with decreased insulin sensitivity [7].

Whenever IR is suspected, blood sample tests are recommended for reliability. Most of the biochemical parameters have been well defined in adults, but appropriate reference data for children are still lacking [8]. Here the researchers used homeostatic model assessment (HOMA) to evaluate insulin sensitivity. HOMA is a method used to quantify IR and beta-cell function and was first described by [9]. The model only requires blood levels of fasting glucose and insulin and is an attempt to demonstrate the relationship between pancreatic insulin production capacity and the ability to maintain adequate glycemic levels [10], [11]. This study aimed to evaluate the relationship between IR, anthropometric indices and biochemical parameters values.

Material and Methods

Subjects

The purposive participants were 97 students with BMI over $\overline{X}$ + 2 SD kg/m^2 from five secondary schools randomized from a total of 18 schools in Mahasarakham province. All participants and their parents understood and consented to enroll in the study and were interviewed one week before the collection of blood about their histories of illness. The research protocol was approved by the ethics committee of Mahasarakham University.

The research design was cross-sectional. Anthropometric measurements, personal data, and venous blood samples were collected from the participants who were aged between 16 and 18 years. Two groups were formed, one (obese group) containing 50 students who had BMI $\geq$ 23 kg/m^2 without additional diseases such as diabetes mellitus, hypertension, and hypothyroidism, the other (normal weight group) a control group of 47 students with BMI < 23 kg/m^2 who had regular health check-ups and no history of obesity in childhood.

Sample size

$$n = \frac{Z^2_{\alpha} PQ}{d^2}$$

$$= \frac{(1.96)^2 (0.5)(0.5)}{(0.10)^2}$$

$$= 96.04 \ (\sim 97)$$

Anthropometrical measurements

Anthropometric parameters included height, weight, biceps, triceps skinfold thickness, and BMI. BMI was calculated by the body weight divided by height squared (kilograms per square meter) and was used to calculate obesity.

Biochemical measurements and tests

Blood samples were taken by professional, medical staff at Mahasarakham Hospital. Blood samples were taken after 12 h of fasting and triglyceride (TG), fasting blood sugar (FBS), and insulin levels were measured. All of the blood samples were immediately processed for TG, FBS, and insulin and the samples were stored at -80°C. TG and FBS were also measured using routine laboratory procedures by Mahasarakham Hospital staff. Serum insulin was measured by a commercial kit assay (LINCO Research, Inc, St. Charles, Missouri, USA). The IR was estimated from fasting serum insulin and serum glucose levels using the HOMA = fasting serum insulin (mU/l) x FBS (mmol/l)/22.5 (Matthews *et al*. 1985).

Statistical methods

The data was analyzed as normal distribution data and expressed as mean and standard deviation. The correlations between HOMA and all parameters were assessed by Pearson Product-moment correlation coefficient. The comparisons of all parameters between the normal weight and obese groups used Independent Sample t-test. All another parameters such as age, FBS, and TG showed a non-

normal distribution (95% C.I). Medians, quartiles, Spearman's rank correlation coefficient (95% C.I) and Mann-Whitney U test were used as non-parametric descriptive statistical methods.

Results

The investigation found a relationship between insulin resistance (IR) with the parameters BMI, biceps skinfold thickness, triceps skinfold thickness, and triglyceride in obese adolescents.

Anthropometric indices and biochemical parameters values

Ninety-seven adolescent participated in this study, 47 with normal weights according to BMI and 50 obese adolescents. Comparisons were made between the two groups found that weights, BMIs, biceps skinfold thicknesses, triceps skinfold thicknesses, and TG and insulin levels in the obese group were significantly higher than students in the normal weights group (p <0.05 for all parameter showed in Table 1). Comparisons of age, height, and FBS were not statistically significantly different in both groups. HOMA in obese participants (0.40 + 0.15) was higher than normal weight (0.37 $\pm$ 0.12) adolescents. FBS were found to be at normal levels, as the participants did not have T2DM (Thai medical DM council; cut-off point < 126 mg/dL; normal group in 83.85 $\pm$ 4.91 mg/dL, obese group in 85.55 $\pm$ 7.47 mg/dL).

Correlation between HOMA and other parameters

Table 2 showed coefficients of simple correlation between HOMA and anthropometry and biochemical parameters in the normal weight and obese groups. In the single linear correlation, for the obese group, HOMA was positively correlated with age, weight, BMI, biceps skinfold thickness and triceps skinfold thickness (p <0.01). Nevertheless some other parameters such as height and TG were not statistically significant for the obese adolescents. Surprisingly age was found to be statistically significantly correlated with HOMA in the obese group but not found in the normal weight group.

Discussion and Conclusion

The rising prevalence of people being overweight is strongly related to the increasing number of cases of impaired glucose regulation in children and adolescents worldwide [11]. Impaired glucose regulation is caused by T2DM [12], [13]. T2DM is considered one of the major metabolic diseases of the 21st century. The excessive intake of food, a sedentary lifestyle, and a lack of physical activity are responsible for the growing epidemic of obesity and the increasing rate of T2DM in many parts of the world [14] Studies indicate relationships between related parameters and IR are present in obese adolescents.

The definition of obesity used in this study was based on BMI and was used to classify the participants into normal weight (nominator for BMI<23 kg/m^2) and obese groups (BMI$\geq$23 kg/m^2). In this study IR status was assessed by HOMA, a simplified tool to estimate insulin sensitivity [15]. HOMA levels were higher in the obese group (0.40 $\pm$ 0.15) while the normal weight group showed HOMA levels lower at 0.37 $\pm$ 0.14. The mean valuesa of HOMA are statistically significantly different. Similar observations were made by several other studies such as Valerio et al. [11] that found HOMA levels higher in an obese group than in a normal weight group.

These results showed that obesity parameters such as BMI, the biceps skinfold thickness and triceps skinfold thickness association with markers of IR had a high statistically significant correlation in obese adolescents (p <0.01). At a given BMI, a high biceps skinfold thickness and triceps skinfold thickness were found to be associated with peripheral obesity. Steven et al. [16] stated that the distribution of body fat could play a role in determination of IR found a relationship between body fat distribution and insulin sensitivity.

These authors said that the central adipose of the human body is a more important determinant of insulin sensitivity than body size alone. The accumulation of body fat centrally is associated with IR, whereas the distribution of body fat in a peripheral pattern is less metabolically important from the standpoint of impairing insulin action. However, it is clear that central adiposity is more important [17], [18] also. Weiss et al. [19] found that overweight individuals with impaired glucose tolerance have peripheral insulin resistance without compensatory insulin secretion and higher visceral and intramuscular fat deposition. Independent of measures of total body fat and ethnicity, abdominal fat deposition is considered a risk factor for IR. In children and adolescents, obesity usually precedes the development of hyperinsulinism that serves to compensate for IR and, thus, prevents the appearance of glucose intolerance or T2DM. Hyperinsulinism stimulates TG accumulation in hepatic and muscle tissues and consequently decreases glucose transporter-4 translocation and favors β-cell apoptosis [20]. The association of obesity with IR and cardiovascular risk is not only related to the degree of obesity but also seems to be critically dependent on body fat distribution. Thus, individuals with greater degrees of central adiposity develop the IR syndrome more frequently than do those with peripheral body fat distribution [21]. Interestingly, Haffner et al. [22] found that visceral adiposity is strongly linked to IR. Therefore, the researchers in this study summarize that BMI, biceps skinfold thickness, and triceps skinfold thickness are related to IR in adolescents.

BMI levels were related to HOMA levels but the age group did not show statistically significant difference only in obese groups. Rosner et al. [23] found that pubertal IR occurs during a time of profound change in body composition and hormone levels. During puberty, BMI increases slowly. Lean body mass and fat mass increase in both sexes but, by the end of puberty, fat accumulation is greater as a percentage of the total body weight in girls than boys. The increase of body fat and BMI correlated strongly with IR and has been proposed as potential mediators of the pubertal changes in IR [24], [5]. However, IR can occur during puberty in the absence of changes in BMI [25]. Moreover, young adult women are more sensitive to insulin than pubertal girls, despite presumably having a greater percentage of body fat [24]. It appears that the factors of changes in body composition may be more important than in the onset of pubertal IR. Thus, in this study, normal weight and obese groups had similar mean ages and no relationship was found between age and IR.

In summary, this study of IR related to anthropometric indices (BMI, biceps skinfold thickness and triceps skinfold thickness). Whereas IR was strongly and similarly related to BMI and adiposity, the results from this study showed that these factors did not completely explain the IR of obese adolescents.

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Table 1. Anthropometric indices and biochemical parameters values.

Parameter	normal-weight (n=47)	obese (n=50)	*p*-value *
	mean ± SD ** (range)	mean ± SD (range)	
Age (years)	17.00 ± 1.00	17.00 ± 0.50	0.614
	(15.00 - 18.00)	(15.00 - 19.00)	
Weight (kg)	51.84 ± 4.80	71.07 ± 11.98	<0.001
	(42.00 - 61.60)	(55.30 - 104.50)	
Height (m)	1.58 ± 0.04	1.60 ± 0.07	0.093
	(1.49 - 1.68)	(1.47 - 1.77)	
BMI (kg/m^2)	20.77 ± 1.44	27.78 ± 3.93	<0.001
	(17.92 – 22.96)	(23.02 – 36.84)	
Biceps skinfold thickness (mm)	14.15 ± 4.86	19.08 ± 8.58	0.001
	(2.56 - 22.16)	(2.50 – 36.16)	
Triceps skinfold thickness (mm)	12.58 ± 4.25	22.43 ± 8.19	<0.001
	(5.93 – 27.00)	(7.66 – 39.33)	
TG (mg/dL)	64.19 ± 17.00	77.00 ± 24.37	0.006
	(30.00 – 289.00)	(33.00 - 299.00)	
FBS (mmol/l)	4.67 ± 0.19	4.67 ± 0.23	0.201
	(4.06 - 5.22)	(4.06 - 6.33)	
Insulin (mU/l)	1.53 ± 0.65	1.87 ± 0.62	0.008
	(0.67 – 2.89)	(0.92 – 3.21)	
HOMA	0.37 ± 0.14	0.40 ± 0.15	0.005
	(0.10 – 0.71)	(0.19 – 0.70)	

* p-value from Independent Sample t-test for all parameters except Age, TG and FBS were from Mann- Whitney U test

** All parameters were used Mean ± SD except Age, FBS, TG were used Median ± QD

Table 2. Pearson's correlation coefficients between HOMA and other parameters.

Parameter	normal-weight (n=47)		obese (n=50)	
	r	*p*-value	r	*p*-value
Age (year)	-0.025	0.727	-0.413	0.003**
Weight (kg)	0.091	0.544	0.468	0.001**
Height (m)	0.020	0.894	0.005	0.974
BMI (kg/m^2)	0.099	0.510	0.555	<0.001**
Biceps skinfold thickness (mm)	-0.059	0.693	0.381	0.006**
Triceps skinfold thickness (mm)	0.047	0.752	0.364	0.009**
TG (mg/dL)	- 0.222	0.134	-0.075	0.602
FBS (mmol/l)	0.354	0.018*	0.518	<0.001**
Insulin (mU/l)	0.992	<0.001**	0.970	<0.001**