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Relation of total homocysteine level with metabolic and anthropometric variables in obese children and adolescents

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Aim: To evaluate the total homocysteine (tHcy) level, a risk factor for atherosclerosis, atherothrombosis, and insulin resistance, for sex and pubertal state differences in obese children. Its relationship with metabolic and anthropometric parameters was also investigated.

Materials and methods: The study involved obese children with a body mass index (BMI) above the 95th percentile who presented with the complaint of excessive weight gain, and healthy children with a BMI below the 85th percentile.

Results: The study included 100 obese (mean age: 10.2 ± 2.7 years) and 71 healthy nonobese (mean age: 10.9 ± 2.6) children. A comparison of the data from the obese group and the control group revealed that the differences in BMI, BMI standard deviation score, tHcy, total cholesterol, triglyceride (TG), insulin, high-density lipoprotein (HDL), low-density lipoprotein (LDL), and homeostasis model assessment-insulin resistance (HOMA-IR) levels were statistically significant ($P < 0.05$). In the obese group, the tHcy level was statistically significantly correlated with age and BMI, TG, and HDL levels ($P < 0.05$), while it was not statistically significantly correlated with total cholesterol, LDL, fasting glucose, insulin, or HOMA-IR levels ($P > 0.05$).

Conclusion: The results of this study showed that the tHcy level was higher in obese children than in healthy children. However, the tHcy level was not significantly correlated with insulin resistance in obese children. Obese children should be routinely screened for high tHcy levels due to the potential atherosclerosis risks, and patients with high tHcy levels should be treated.

Key words: Atherosclerosis, homocysteine, insulin resistance, obesity, puberty

Obez çocuklar ve ergenlerde total homosistein düzeyinin metabolik ve antropometrik değişkenler ile olan ilişkisi

Amaç: Obez çocuklar için ateroskleroz, aterotromboz ve insülin direnci gelişimi için risk faktörü olan total homosistein (tHcy) düzeyini cinsiyet ve pubertal durum farklılığına göre değerlendirerek, metabolik ve antropometrik parametreler ile olan ilişkisini araştırmak amaçlanmıştır.

Yöntem ve gereç: Çalışmaya, kilo artışı yakınması ile başvuran vücut kitle indeks (VKİ) persentili 95'in üzerinde olan obez çocuklar ve VKİ persentili 85'in altında olan sağlıklı çocuklar alındı.

Bulgular: Çalışmaya toplam 100 obez (ortalama yaş: 10,2 ± 2,7 yıl) ve 71 sağlıklı obez olmayan (10,9 ± 2,6) çocuk alındı. Obez grubun verileri kontrol grup ile karşılaştırıldığında VKİ, VKİ-standart sapma skoru, tHcy, total kolesterol, trigliserit (TG), insülin, yüksek dansiteli lipoprotein (HDL), düşük dansiteli lipoprotein (LDL) ve homeostasis model assessment-insülin direnci (HOMA-IR) düzeyleri arasında istatistiksel olarak anlamlı fark saptandı ($P < 0,05$). Obez

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grupta tHcy düzeyi puberte ve cinsiyet durumuna göre karşılaştırıldığında pubertedeki ve erkek hasta grubunda istatistiksel olarak anlamlı yüksek saptandı (P < 0,05). Obez grupta tHcy düzeyi yaş, VKİ, TG ve HDL düzeyleri ile istatistiksel olarak anlamlı korelasyon gösterdiği saptanırken (P < 0,05), total kolesterol, LDL, açlık glukoz, insülin ve HOMA-IR düzeyi ile anlamlı korelasyon göstermediği bulundu (P > 0,05).

Sonuç: Bu çalışmanın sonuçları tHcy düzeyinin obez çocuklarda sağlıklı çocuklara göre daha yüksek olduğunu göstermiştir. Buna karşın, tHcy düzeyinin obez çocuklarda insülin direnci ile anlamlı korelasyonunun olmadığı bulundu. Obez çocuklarda tHcy düzeyi ateroskleroz riski nedeniyle rutin olarak taranmalıdır ve yüksek tHcy düzeyi olan hastalar tedavi edilmelidir.

Anahtar sözcükler: Ateroskleroz, homosistein, insülin direnci, obezite, puberte

Introduction

Obesity is a chronic metabolic disease associated with cardiovascular and atherosclerotic changes. Its incidence among children and adolescents has increased 3-fold in the past 20 years (1,2). In many studies, obesity has been shown to cause hypertension, dyslipidemia, type 2 diabetes mellitus, and cancer (2,3).

The complications associated with obesity pave the way for the development of atherosclerotic pathologies. In obese individuals, various laboratory anomalies (sensitive C-reactive protein, interleukin-6, tumor necrosis factor, lipid profile, and adiponectin, among others) and clinical parameters (hypertension, intima-media thickness, hypertrophy of the left ventricle, vascular function disorders, and microalbuminuria) have been identified as atherosclerosis markers (4-7). Recent studies, particularly on adults, have suggested that elevations in total homocysteine (tHcy) levels are important markers for the development of atherothrombosis and atherosclerotic changes (8-13). Furthermore, tHcy has been found to be significantly correlated with body mass index (BMI) and insulin resistance (IR); it also contributes to the development of IR (14-16).

The onset of atherosclerotic change is in its infancy in obese children, and, in recent years, it has been suggested that tHcy is one of the risk factors (7,13,16,17). However, data on obese children are not definitive and have remained controversial. Thus, this study evaluated tHcy, a risk factor for atherosclerosis, atherothrombosis, and IR, according to sex and pubertal state differences in obese children. In addition, the relationship of tHcy with metabolic (lipid profile, glucose level, insulin level, and IR index) and anthropometric parameters (BMI and BMI standard deviation score (SDS)) was investigated.

Materials and methods

The study included obese children and adolescents with a BMI above the 95th percentile, as according to data from the Centers for Disease Control and Prevention (CDC), who applied to the Keçiören Training and Research Hospital complaining of weight gain, and healthy children and adolescents with a BMI below the 85th percentile who were similar in age and sex distribution to the children in the obese group. For calculations of BMI-SDS, data from the CDC were used (18).

Before the outset of the study, all of the patients and control subjects were given thorough physical examinations and laboratory tests (thyroid function test and cortisol measurement) for potential endocrine pathology. Those with chronic diseases that could cause obesity (cardiovascular, gastrointestinal, and respiratory), a history of drug use (steroids and antipsychotics), endocrine pathology (Cushing syndrome and hypothyroidism), or suspected syndromes associated with obesity (Prader-Willi and Laurence-Moon-Biedle syndromes) were excluded from the study.

Patient height was measured using a Harpenden stadiometer with a sensitivity of 0.1 cm, and weight was measured using a seca scale with a sensitivity of 0.1 kg. The weight of each subject was measured with all clothing removed except undergarments. BMI was calculated by dividing weight (kg) by height squared (m^2). Findings for pubertal development were evaluated according to Tanner staging. A testes volume of ≥4 mL in males and breast growth of stage

2 and over (Tanner) in females were considered to be findings of pubertal development.

The blood samples were obtained intravenously after at least 12 h of fasting. Fasting serum glucose level was measured using an automated analyzer (Konelab 60I, Thermo Scientific, Finland) by the enzymatic method (Lot No. D426, Konelab). Fasting insulin level was measured by the immunoluminometric method (DiaSorin). Total cholesterol (TC) (Lot No. B540, Konelab) and triglyceride (TG) (Lot No. C186, Konelab) levels were measured by the enzymatic calorimetric method. Low-density lipoprotein cholesterol (LDL-C) (Lot No. C435, Konelab) and high-density lipoprotein cholesterol (HDL-C) (Lot No. C136, Konelab) levels were measured by the homogenous enzymatic method. Plasma tHcy analysis was performed using an AXSYM device (Abbott Laboratories, Medical Diagnostics Products, NJ, USA) and the Abbott Homocysteine Kit with the macro-enzyme linked immunosorbent assay (macro-ELISA) method (Abbott Laboratories). The normal value for tHcy levels was considered to be 3.3-11.3 μmol/L. Cutoff points above the 95th percentile of healthy children were used to define dyslipidemia and impaired fasting glucose, according to international recommendations (19,20). IR was evaluated according to the homeostasis model assessment-insulin resistance (HOMA-IR) index. Different cutoff values for prepubertal (HOMA-IR > 2.5) and pubertal (HOMA-IR > 4.0) stages were used to determine the status of IR (21).

The statistical analyses of the data were conducted with SPSS 16.0.1 (SPSS Inc., Chicago, IL, USA). All values were presented as mean ± SD. The distribution of data was evaluated with the Kolmogorov-Smirnov test. For comparisons of the groups (obese and control; insulin-resistant and nonresistant), Student's t-test was used. The correlation between the independent parameters was investigated with Pearson's correlation analysis. $P < 0.05$ was considered statistically significant.

The study was initiated upon approval of the local ethics committee of Keçiören Training and Research Hospital in light of the Helsinki Declaration. The written informed consent of the parent(s) of each subject was also obtained before the study.

Results

The study included 100 obese (mean age: 10.2 ± 2.7 years; 49 male, 41 pubertal) and 71 healthy nonobese (mean age: 10.9 ± 2.6 years; 36 male, 35 pubertal) children. A comparison of the data from the obese group and the control group revealed that the differences between the BMI, BMI-SDS, tHcy, TC, TG, insulin, HDL-C, LDL-C, fasting glucose, and HOMA-IR levels of the 2 groups were statistically significant ($P < 0.05$) (Table 1).

In the obese group, 34 (34%) children had an elevated tHcy level, while in the control group, 9 (12.6%) children had an elevated tHcy level.

In the obese group, tHcy levels were compared according to pubertal stage and sex and were found to be statistically significantly higher in the pubertal and male patients (12.8 ± 6.5 and 9.4 ± 2.9, $P = 0.019$; 12.6 ± 5.9 and 9.0 ± 2.9, $P = 0.001$, respectively). However, in the control group, no differences were found between the tHcy levels of pubertal and prepubertal or between male and female subjects (10.1 ± 2.6 and 8.7 ± 3.4, $P = 0.065$; 9.1 ± 2.8 and 9.7 ± 3.4, $P = 0.382$, respectively) (Tables 2 and 3).

In the obese group, the ages of males and those in the pubertal stage were higher than those of the females and the subjects in the prepubertal stage (10.8 ± 2.5 and 9.6 ± 2.7 years, $P = 0.039$; 12.4 ± 1.7 and 8.7 ± 2.2 years, $P = 0.001$, respectively).

In the comparisons of the obese cases for insulin levels, no statistical differences were found for tHcy levels ($P > 0.05$). In the obese group, the tHcy level was statistically significantly correlated with age, BMI, and TG and HDL-C levels ($P < 0.05$), while it was not statistically significantly correlated with TC, LDL-C, fasting glucose, insulin, or HOMA-IR levels ($P > 0.05$) (Table 5).

No statistically significant differences were found between the tHcy levels and ages of prepubertal obese patients and prepubertal healthy children ($P > 0.05$), while a statistically significant difference was found between the 2 groups for IR and BMI ($P = 0.05$) (Table 2). On the other hand, no statistically significant age difference was noted between pubertal obese children and pubertal healthy children ($P > 0.05$), whereas statistically significant differences

Table 1. The clinical and laboratory characteristics of the obese and control groups (mean ± SD).

Characteristics	Obese group (n = 100)	Control group (n = 70)	P-value[a]
Age (years)	10.2 ± 2.7	10.8 ± 2.6	0.136
BMI (kg/m²)	27.6 ± 4.2	17.7 ± 2.3	**0.001**
BMI-SDS	2.2 ± 0.4	0.1 ± 0.7	**0.001**
tHcy (µmol/L)	10.8 ± 5.0	9.4 ± 3.1	**0.041**
TC (mg/dL)	162.5 ± 28.9	152.6 ± 25.3	**0.022**
Triglyceride (mg/dL)	129.7 ± 59.4	91.6 ± 50.7	**0.001**
HDL-C (mg/dL)	44.1 ± 9.9	56.0 ± 17.1	**0.001**
LDL-C (mg/dL)	93.5 ± 24.9	85.2 ± 17.6	**0.017**
Glucose (mg/dL)	89.6 ± 7.0	90.8 ± 7.6	0.306
Insulin (IU/mL)	16.0 ± 9.3	9.6 ± 5.7	**0.001**
HOMA-IR	3.6 ± 2.2	2.1 ± 1.28	**0.001**

[a]Student's t-test.
BMI: body mass index, BMI-SDS: standard deviation score of body mass index, tHcy: total homocysteine, HDL-C: high density lipoprotein-cholesterol, HOMA-IR: homeostasis model assessment-insulin resistance, LDL-C: low density lipoprotein-cholesterol, TC: total cholesterol.

Table 2. The laboratory and anthropometric characteristics of the obese and control groups according to pubertal status (mean ± SD).

	Prepubertal (obese: n = 59, control: n = 36)				Pubertal (obese: n = 41, control: n = 35)			
	tHcy	**Age**	**HOMA-IR**	**BMI**	**tHcy**	**Age**	**HOMA-IR**	**BMI**
Obese	9.4 ± 2.9[¥]	8.7 ± 2.9[&]	3.0 ± 2.0[£]	26.1 ± 3.5[∞]	12.8 ± 6.5[¥]	12.4 ± 1.7[&]	4.5 ± 2.8[£]	29.8 ± 4.1[∞]
Control	8.7 ± 3.4	9.1 ± 1.8[β]	1.6 ± 1.0[α]	16.9 ± 2.3[€]	10.1 ± 2.6	12.6 ± 2.0[β]	2.6 ± 1.3[α]	18.5 ± 2.1[€]
P*	0.300	0.344	**0.001**	**0.001**	**0.027**	0.633	**0.001**	**0.001**

*, ¥, £, ∞, β, α, €: P < 0.05 (Student's t-test).
BMI: body mass index, tHcy: total homocysteine, HOMA-IR: homeostasis model assessment-insulin resistance.

were found between the tHcy, IR, and BMI values of the 2 groups (P < 0.05) (Table 2).

The tHcy levels, ages, and HOMA-IR and BMI results of the pubertal obese patients and prepubertal obese patients were statistically significantly different (P < 0.05); however, comparisons of the pubertal and prepubertal children in the control group showed no differences between the tHcy levels. The ages and the IR and BMI results of these groups (pubertal and prepubertal controls) were statistically significantly different (P < 0.05) (Table 2).

In 47 (47%) of obese patients and in 11 (15.5%) of the control subjects, IR was detected. Comparisons of the children in the obese group for HOMA-IR presence showed that there were statistically significant differences between the age and BMI values of the children with HOMA-IR and the children without HOMA-IR; however, no statistically significant difference was found between the groups for tHcy levels (11.1 ± 4.1 and 10.5 ± 5.6, P = 0.60, respectively) (Table 4).

While in the obese group, the tHcy level was significantly correlated with age, BMI, and TG and HDL-C levels (P < 0.05), no correlations were determined between tHcy and TC, LDL-C, fasting glucose, insulin, and HOMA-IR levels (P > 0.05). In

Table 3. The clinical and laboratory characteristics of the obese and control groups according to sex (mean ± SD).

Characteristics	Obese group			Control group		
	Male (n = 49)	Female (n = 51)	p[a]	Male (n = 36)	Female (n = 35)	p[a]
Age (years)	10.8 ± 2.5	9.6 ± 2.7	0.039	11.0 ± 2.6	10.6 ± 2.5	0.452
BMI (kg/m²)	28.5 ± 3.9	26.8 ± 4.2	0.181	17.9 ± 2.2	17.5 ± 2.4	0.464
BMI-SDS	2.3 ± 0.5	2.2 ± 0.4	0.230	0.07 ± 0.7	–0.1 ± 0.9	0.467
tHcy (µmol/L)	12.6 ± 5.9	9.0 ± 2.9	0.001	9.1 ± 2.8	9.7 ± 3.4	0.382
TC (mg/dL)	162 ± 28	162 ± 29	0.956	156 ± 24	148 ± 25	0.166
Triglyceride (mg/dL)	141 ± 62	118 ± 54	0.047	84 ± 52	99 ± 48	0.198
HDL-C (mg/dL)	42 ± 9	45 ± 10	0.069	55 ± 12	56 ± 20	0.853
LDL-C (mg/dL)	94 ± 23	92 ± 26	0.734	87 ± 18	83 ± 17	0.334
Glucose (mg/dL)	90.5 ± 6.8	88.8 ± 7.2	0.227	92.5 ± 7.2	89 ± 7.7	0.053
Insulin (IU/mL)	16.0 ± 9.4	15.1 ± 9.15	0.326	9.3 ± 5.7	9.7 ± 5.8	0.680
HOMA-IR	3.9 ± 2.3	3.3 ± 2.0	0.256	2.1 ± 1.3	2.1 ± 1.2	0.965

[a]Student's t-test.
BMI: body mass index, BMI-SDS: standard deviation score of body mass index, tHcy: total homocysteine, HDL-C: high density lipoprotein-cholesterol, HOMA-IR: homeostasis model assessment-insulin resistance, LDL-C: low density lipoprotein-cholesterol, TC: total cholesterol.

Table 4. The clinical and laboratory characteristics of the obese group with and without insulin resistance (mean ± SD).

	With insulin resistance (n = 47)	Without insulin resistance (n = 53)	p[a]
Age (years)	11.0 ± 2.5	9.6 ± 2.7	**0.006**
BMI (kg/m²)	29.2 ± 3.83	26.2 ± 4.0	**0.001**
BMI-SDS	2.28 ± 0.4	2.17 ± 0.5	0.230
tHcy (µmol/L)	11.1 ± 4.1	10.54 ± 5.6	0.600
TC (mg/dL)	167.5 ± 28.5	157.9 ± 28.8	0.096
Triglyceride (mg/dL)	142.5 ± 62.6	118.2 ± 54.5	**0.041**
HDL-C (mg/dL)	44.8 ± 9.5	46.2 ± 9.9	**0.026**
LDL-C (mg/dL)	98.9 ± 23.4	88.8 ± 25.5	**0.042**
Glucose (mg/dL)	90.6 ± 8.0	88.8 ± 5.9	0.193
Insulin (IU/mL)	16.0 ± 9.3	9.6 ± 5.7	**0.001**
HOMA-IR	5.3 ± 2.1	2.1 ± 0.9	**0.001**

[a]Student's t-test.
BMI: body mass index, BMI-SDS: standard deviation score of body mass index, tHcy: total homocysteine, HDL-C: high density lipoprotein-cholesterol, HOMA-IR: homeostasis model assessment-insulin resistance, LDL-C: low density lipoprotein-cholesterol, TC: total cholesterol.

Table 5. The correlations between total homocysteine level and clinical laboratory parameters of the obese and control groups.

	Obese group		Control group	
	r	P[a]	r	P[a]
Age (years)	0.449	**0.001**	0.340	**0.040**
BMI (kg/m²)	0.374	**0.001**	0.138	0.258
BMI-SDS	−0.028	0.782	−0.085	0.479
Triglyceride (mg/dL)	0.293	**0.003**	−0.103	0.397
TC (mg/dL)	−0.089	0.381	−0.126	0.296
LDL-C (mg/dL)	−0.081	0.421	−0.226	0.058
HDL-C (mg/dL)	−0.289	**0.040**	−0.027	0.825
Glucose (mg/dL)	−0.163	0.105	−0.168	0.162
Insulin (IU/mL)	0.188	0.061	−0.041	0.733
HOMA-IR	0.150	0.138	−0.017	0.888

[a]Pearson's correlation analysis.
BMI: body mass index, BMI-SDS: standard deviation score of body mass index, tHcy: total homocysteine, HDL-C: high density lipoprotein-cholesterol, HOMA-IR: homeostasis model assessment-insulin resistance, LDL-C: low density lipoprotein-cholesterol, TC: total cholesterol.

the control group, the tHcy level was only significantly correlated with age ($P < 0.05$) (Table 5).

Discussion

Homocysteine is normally a sulfur amino acid that is formed by the transmethylation of methionine amino acid. It can retransform into methionine amino acid by remethylation, accompanied by folate and vitamin B12, as well as into cysteine amino acid by cystathionine-β-synthase enzyme mediated with vitamin B6. The pathology that develops due to cystathionine-β-synthase enzyme deficiency is associated with premature atherosclerosis and recurrent thromboembolic events in homocystinuria. In addition, nutritional (folic acid and vitamins B6 and B12), genetic (methylenetetrahydrofolate reductase gene mutations), and endocrine factors, as well as cancer, human immunodeficiency virus, and renal failure, have been claimed as conditions responsible for moderately high levels of tHcy (22,23). Childhood obesity, accompanied by nutritional deficiencies and endocrine disorders, is one of the important chronic metabolic diseases. In recent years, controversial results regarding the association between obesity and tHcy level have been reported. Moreover, tHcy level has been reported to vary according to age, sex, and pubertal status in healthy, obese, and diabetic children (22,24,25).

Brasileiro et al., in their study on overweight and healthy pubertal children, found no statistically significant differences between the tHcy levels of 86 overweight and 153 healthy children (24). Papandreou et al. evaluated school children, 6-15 years of age, and determined no statistically significant differences between the tHcy levels of obese (n = 41), overweight (n = 102), and healthy (n = 381) children regardless of their pubertal stage (26). Similarly, Martos et al. did not find any statistically significant difference between the tHcy levels of prepubertal obese children (n = 43) and nonobese children (n = 43) who were 6-9 years of age (13). On the other hand, in a study by Ustundag et al., the tHcy levels of 60 prepubertal obese children were statistically higher than those of the 60 children in the control group (27). Likewise, Narin et al. reported significantly higher levels of tHcy in obese children than in the control group (aged 7-17 years), regardless of pubertal stage (28). In our study, the tHcy levels of obese children were statistically higher than those of healthy children.

In the literature, tHcy level has been reported to vary depending on age, sex, IR, and pubertal status (22,24,25). It has also been shown to positively

correlate with age and sex in healthy children (22,24). In their study on healthy children, De Laet et al. determined that the tHcy level of the adolescents, 15-19 years of age, was higher than that of the children, 5-9 years of age (22). In the same study, a comparison by sex of healthy children, 15-19 years of age, showed that the tHcy level was statistically significantly higher in the males than in the females. Brasileiro et al. determined a correlation between tHcy level and age and sex in the control subjects and overweight children (24). In our study, the tHcy level was significantly correlated with age, in both the control and obese groups, and the male subjects had significantly higher tHcy levels than female subjects in the obese group.

Although no differences in tHcy levels were reported in obese children for different pubertal stages, in a study of type I diabetes mellitus patients, the level of tHcy was significantly correlated with pubertal stage (25). In addition to a lack of difference by age, in our study we found that tHcy levels of children in the prepubertal obese and control groups were not statistically significantly different, while tHcy levels of children in the pubertal obese and control groups were statistically significantly different. Moreover, in the comparison of the tHcy levels of the prepubertal obese and control groups, the tHcy levels of the control group did not differ in a statistically significant manner according to age, IR, or BMI. In the obese pubertal patients, however, the tHcy levels were statistically significantly higher than those of the prepubertal obese patients. These results support the finding that, in addition to the factors that affect tHcy level, such as age, BMI, and IR, nutritional status in pubertal obese patients is more disordered than in the prepubertal obese patients.

Obesity is one of the important markers of unhealthy and imbalanced nutrition. Folate and vitamin B12 deficiencies have been reported to be the most important causes of elevated tHcy levels (24,29). Brasileiro et al. determined folate deficiency in 68.6% of overweight children with elevated tHcy levels and emphasized deficiencies in vitamin B12 and folate, which are usually supplied in the daily diet (24). Lin et al. showed a significant negative correlation between folate and vitamin B12 level and tHcy level (29). However, in our study, the dietary habits of the patients were not investigated in detail, and vitamin B12 and folate levels were not studied; this is a drawback of the current study.

In the literature, tHcy level has been reported to be associated with insulin levels in obese individuals (13). Nevertheless, whether IR leads to increases in tHcy levels or elevated tHcy levels lead to IR remains controversial (14). In a study by Martos et al. of children of 6-9 years of age, the tHcy levels of hyperinsulinemic obese subjects (n = 14) were significantly higher than levels in normoinsulinemic obese subjects (n = 29) and were shown to be significantly correlated with fasting glucose and HOMA-IR levels (13). In our study, which had a larger number of participants, the tHcy levels of the obese patients with IR (n = 47) were higher than those of the obese patients without IR (n = 53); however, the difference was not statistically significant. Moreover, in the obese group, the tHcy level was not significantly correlated with HOMA-IR or fasting glucose levels. These findings show that there is no significant correlation between tHcy level and IR.

The results of this study show that the tHcy levels of obese and healthy children differ depending on age, sex, and pubertal status, but that these levels are not correlated with IR. In addition, the tHcy levels of obese adolescents in the pubertal stage are higher than the levels in prepubertal obese children. Obese children should be routinely screened for high tHcy levels due to the potential risk of atherosclerosis, and patients with high tHcy levels should be treated to prevent prospective vascular pathologies.

Conclusion

Based on the findings of this study, it can be emphasized that, to prevent potential cardiovascular diseases in pubertal (adolescent) obese children and prepubertal obese children, the dietary habits and nutritional intake of these children should be improved according to the needs of the age group, and preventive health measures should be taken to avoid or overcome obesity.

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