Clin Nutr Res. 2022 Jul;11(3):194-203
https://doi.org/10.7762/cnr.2022.11.3.194
pISSN 2287-3732·eISSN 2287-3740

cnr
CLINICAL NUTRITION RESEARCH

Original Article

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Evaluation of Serum Omentin-1, Vaspin, Leptin, Adiponectin Levels in Obese/Overweight Children and Their Relationship With Non-Alcoholic Fatty Liver Disease

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OPEN ACCESS

Received: May 30, 2022
Revised: Jul 6, 2022
Accepted: Jul 11, 2022
Published online: Jul 25, 2022

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Funding
The article was supported by the Yozgat Bozok University scientific research project.

Conflict of Interest
The authors declare that they have no competing interests.

ABSTRACT

To investigate adipokines (vaspin, omentin-1, adiponectin and leptin) and their correlation with hepatosteatosis degree in obese/overweight (O/O) children. We analyzed adipokine levels of 81 children (49 O/O, [body mass index (BMI) > 95th] and 32 non-obese (BMI = 5-85th) admitted to the pediatric outpatient clinic. Serum triglyceride, glucose, low density lipoprotein-cholesterol, total cholesterol, high density lipoprotein-cholesterol, alanine aminotransferase, aspartate aminotransferase (AST), insulin, HbA1c levels and leptin, omentin-1, vaspin, adiponectin levels were studied. O/O children with hepatosteatosis were divided into grades 1, 2 and 3 according to the degree of hepatosteatosis determined by ultrasonography. While AST (p = 0.001), triglyceride (p = 0.006), BMI percentile (p = 0.000), HOMA index (p = 0.002), systolic blood pressure (p = 0.02), leptin (p = 0.001), omentin-1 (p = 0.001), adiponectin (p = 0.001) levels were higher, vaspin level was lower (p = 0.008) in the (O/O) group compared to the controls. There was a positive correlation between HDL and vaspin, and a negative correlation between HDL and omentin-1 in the O/O group. Also it was observed that as the degree of hepatosteotosis increased, leptin (p = 0.004), omentin-1 (p = 0.001) levels were increased. There was no significant change in vaspin level (p = 0.128). The high levels of omentin-1, leptin and adiponectin have an association with the development of hepatosteatosis in O/O children.

Keywords: Overweight; Children; Adipokines; Nonalcoholic fatty liver disease

INTRODUCTION

The global epidemic of childhood obesity, which accompanies the increase in the prevalence of cardiovascular, metabolic and endocrine comorbidities in young people, is one of the world's most important public health problems. The increasing prevalence of pediatric obesity leads to a potential decrease in life expectancy [1-4].

Non-alcoholic fatty liver disease (NAFLD) is a liver disorder characterized by the accumulation of triglycerides within hepatocytes [5]. This excessive storage of fat in the liver

Author Contributions

Conceptualization:Akyüz Özkan E; Data curation: Sadigov A; Formal analysis: Akyüz Özkan E, Sadigov A, Öztürk O; Investigation: Akyüz Özkan E, Sadigov A; Methodology: Akyüz Özkan E, Sadigov A; Writing - original draft: Akyüz Özkan E, Sadigov A; Writing - review & editing: Akyüz Özkan E, Sadigov A,Öztürk O.

makes the organ susceptible to inflammation and fibrosis. NAFLD is strongly associated with obesity, type 2 diabetes mellitus (T2DM), and cardiovascular disease [6].

It has been shown that adipose tissue secretes many bioactive agents and that explains the relationship between increased metabolic risk and visceral fat mass. These agents, called adipokines, include vaspin, leptin, adiponectin, and omentin-1, play an important role in interactions between various systems, such as central and peripheral nervous, immune and adrenal system [3].

Although the debilitating effect of leptin is well known, serum leptin levels are high in obese children and are closely related to the severity of obesity. In most cases of obesity, leptin does not cause weight loss, despite both high circulating leptin levels and an intact leptin receptor. This is explained by the presence of leptin resistance in obese individuals. The positive association of leptin with body weight and especially body mass index (BMI) has been demonstrated in all age groups, including newborns and those undergoing puberty [4].

Vaspin, a protein from the serine protease inhibitor family, is expressed in visceral adipose tissue. Studies have shown that serum vaspin levels increase in type 2 diabetes, obesity and impaired insulin sensitivity, but decrease with regression of diabetes, weight loss and increase in insulin sensitivity. After metformin treatment, there was a remarkable decrease in serum vaspin levels [7,8].

In obese individuals, both the gene expression and plasma levels of omentin-1 in adipose tissue are decreased. Obesity-related markers such as BMI, waist circumference, and leptin levels are inversely related to omentin-1 levels. Serum omentin-1 levels were found to be lower in patients with obesity-related conditions such as impaired glucose tolerance, polycystic ovary syndrome or T2DM, compared to the normal population. Following weight loss with diet, there is an increase in insulin sensitivity and serum omentin-1 levels [9]. Metformin, a drug that increases insulin sensitivity, increases omentin-1 levels. Plasma omentin-1 levels are negatively correlated with HOMA index and positively correlated with plasma high density lipoprotein (HDL) and adiponectin levels [9].

Serum concentration of adiponectin is decreased in patients with obesity and insulin resistance. Adiponectin is a molecule that increases insulin sensitivity and fatty acid oxidation. Adiponectin enhances the effects of insulin in the liver, increases insulin sensitivity and decreases hepatic glucose production [10]. It has been reported in the literature that the risk of developing type 2 diabetes in individuals with high adiponectin levels were lower than those with low adiponectin levels. Plasma adiponectin levels increase in conditions that increase insulin sensitivity, such as weight loss, diet, and thiazolidinedione therapy [11].

There is evidence that several adipokines play an important role in the pathogenesis of NAFLD and may be responsible for fat accumulation and insulin resistance [12]. The interaction of various cytokines derived from adipose tissue may play a crucial role in the progression of the disease from fatty infiltration to inflammation.

NAFLD includes a broad spectrum of liver cell damage induced by insulin resistance. Primarily, fat deposition in hepatocytes (simple fatty liver) occurs as a result of hepatic insulin resistance. The adipokines are thought to modulate these metabolic processes by

https://doi.org/10.7762/cnr.2022.11.3.194

regulating insulin-mediated glucose metabolism, fatty acid utilization, and lipid deposition of visceral tissues. In the later stages, inflammatory phenomena occur, which can progress to steatohepatitis and eventually cirrhosis. It has been suggested that the development of steatohepatitis is a result of the balance between the pro- and anti-inflammatory effects of adipokines [13]

In this study, our aim was to measure circulating levels of leptin, omentin-1, vaspin and adinopectin in obese/overweight (O/O) children and makes a comparison with age-matched controls. We also analyzed the correlations between these adipokines and laboratory markers and the degree of fatty liver.

MATERIALS AND METHODS

This observational, prospective study consisted of 81 children admitted to the pediatric outpatient clinic between August 2019 and December 2019. Ethics committee approval was obtained for the study from the Institutional Review Board. (2017-KAEK-189_2018.03.21_10). Consent forms were obtained from all children included in the study or their parents, explaining the purpose and characteristics of the study.

Children under the age of 18 who were obese or overweight according to BMI percentile, included in the study. Children were excluded if they had a major illness including T1DM or T2DM, or had a condition known to influence body composition, took medications, or insulin action, or insulin secretion (e.g. glucocorticoid therapy, hypothyroidism, Cushing's disease). Patients with viral hepatitis, hemochromatosis, Wilson disease, autoimmune hepatitis, primary biliary cirrhosis, sclerosing cholangitis, biliary obstruction, α1 antitrypsin deficiency, ischemic cardiac or cerebrovascular disease, impaired renal function, or malignancies were also excluded from the present study. All subjects included in the control group were judged to be in good health, with normal results on liver function tests.

After measuring the height and weight of the children, BMI was calculated with the formula (weight [kg]/height [m^2]) and BMI percentile was calculated. Children with a BMI between the 85th and 95th percentile were considered as overweight, and those over 95th were considered as obese, between 5-85th percentile was considered as normal weight children in line with the Centers for Disease Control and Prevention (CDC) recommendation [14].

After the measurements were taken, at least 8 hours of fasting blood samples were taken from all children. Serum glucose, low density lipoprotein-cholesterol, total cholesterol, HDL--cholesterol, triglyceride, aspartate aminotransferase (AST), alanine aminotransferase (ALT), fasting insulin, glucose, HbA1c levels were studied from serum samples obtained by centrifuging blood samples at 3,000 rpm for 10 minutes within the first hour in the Yozgat Bozok University Hospital Laboratory. SunRed brand Elisa kits were used for measuring leptin, omentin-1, vaspin, adiponectin levels.

After the results were obtained, HOMA-IR was calculated with the formula: Fasting insulin (μU/mL) × fasting glucose (mg/dL)/405. Abdominal ultrasonography was performed to evaluate hepatosteatosis degree in O/O children included in the study. The diagnosis of NAFLD was based on increased echogenicity determined by ultrasound compatible with fatty infiltration of the liver with or without elevated ALT levels.

https://doi.org/10.7762/cnr.2022.11.3.194

Statistical analysis

Statistical analyses were performed with the SPSS 20.0 package program (IBM Corp., Armonk, NY, USA). Non-parametric and parametric tests were used for the assessment of all results. Kolmogorov Smirnov test was used to determine whether the distributions were normally distributed. In addition, T-test, Kruskal Wallis, Mann Whitney U and χ^2 tests were used to evaluate the groups. Significance levels were accepted as $p < 0.05$.

RESULTS

A total of 81 children, 49 O/O (19 male, 30 female, mean age; 13.18 ± 2.48 years) and 32 non-obese (13 male and 19 female, mean age; 11.97 ± 3.50 years), were included in the study. Of the 49 O/O children, 9 were in overweight, 40 were in obese group.

While AST (p = 0.001), triglyceride (p = 0.006), HOMA index (p = 0.002), systolic blood pressure (p = 0.002), leptin (p = 0.001), omentin-1 (p = 0.001), adiponectin (p = 0.001), BMI percentile (p = 0.000) levels were higher, vaspin level was lower (p = 0.008) in the O/O group compared to the controls. There was no significant difference between other values (**Table 1**). No statistical significance was found on comparing serum adipokine levels with gender.

There was a positive correlation between HDL and vaspin, and a negative correlation between HDL and omentin-1 in the O/O group (**Table 2**).

A total of 22 children had grade 1 (12 female, 10 male), 9 had grade 2 (3 female, 6 male) and 1 boy had grade 3 hepatosteatosis. The severity of hepatosteatosis was higher in males. It was observed that HbA1c increased as the degree of hepatosteatosis increased (p = 0.036). There was no significant difference in other laboratory values between children with hepotosteatosis (**Table 3**).

Table 1. Demographic features and laboratory data of the control and obese group

Variables	Control group (mean ± SD) (n = 32)	Obese/Overweight group (mean ± SD) (n = 49)	p value
Age (yr)	11.97 ± 3.50	13.18 ± 2.48	0.094*
Male/female	13/19	19/30	0.0124†
Systolic BP (mmHg)	98.26 ± 9.69	105.0 ± 10.0	**0.002**
Diastolic BP (mmHg)	60.69 ± 7.68	63.95 ± 8.76	0.071
BMI percentile	42.72 ± 28.96	96.10 ± 4.58	**0.000**
AST (U/L)	14.32 ± 5.76	27.95 ± 34.49	**0.001***
ALT (U/L)	22.41 ± 7.70	24.23 ± 14.95	0.478*
Total cholesterol (mg/dL)	147.82 ± 24.21	148.95 ± 22.71	0.841*
Triglyceride (mg/dL)	87.21 ± 35.38	120.50 ± 70.13	**0.006***
HOMA index	2.59 ± 1.92	6.51 ± 7.90	**0.002***
Glucose (mg/dL)	93.21 ± 10.92	95.06 ± 9.62	0.440*
HBA1c (%)	5.32 ± 0.29	5.39 ± 0.28	0.276
HDL (mg/dL)	45.18 ± 9.14	41.72 ± 9.40	0.115*
LDL (mg/dL)	78.67 ± 17.40	80.32 ± 23.14	0.770*
Leptin (ng/mL)	12.49 ± 2.92	15.52 ± 3.13	**0.001***
Omentin-1 (pg/mL)	197.49 ± 37.45	244.59 ± 33.61	**0.001***
Vaspin (pg/mL)	1,355.30 ± 280.90	1,203.94 ± 155.71	**0.008***
Adiponectin (μg/mL)	10.44 ± 2.05	13.49 ± 2.66	**0.001***

SD, standard deviation; BP, blood pressure; BMI, body mass index; AST, aspartate aminotransferase; ALT, alanine aminotransferase; HDL, high density lipoprotein; LDL, low density lipoprotein.
*Student's t-test, †χ^2 test, Mann-Whitney U test, bold indicates statistically significant ($p < 0.05$).

https://doi.org/10.7762/cnr.2022.11.3.194

Table 2. Correlations of laboratory values in the obese/overweight group

Obese/overweight group (n = 49)	AST (U/L)	ALT (U/L)	T.cholesterol (mg/dL)	Triglyceride (mg/dL)	HOMA index	Glucose (mg/dL)	HbA1C (%)	HDL cholesterol (mg/dL)	LDL cholesterol (mg/dL)	BMI per.	Leptin (ng/mL)	Omentin-1 (pg/mL)	Vaspin (pg/mL)	Adiponectin (μg/mL)
Age (yr)	0.001	0.172	0.085	0.156	0.035	0.073	0.049	0.020	0.011	**0.230***	0.116	−0.121	0.147	**0.433***
AST (U/L)	1	**0.898**†	−0.002	0.067	−0.082	−0.104	−0.072	−0.150	0.057	0.080	−0.142	0.168	0.011	−0.107
ALT (U/L)		1	−0.030	0.153	−0.014	−0.142	−0.048	−0.192	0.014	**0.238***	−0.097	0.255	−0.055	−0.144
Total cholesterol (mg/dL)			1	0.210	−0.079	−0.128	0.040	**0.319**†	**0.864**†	−0.097	0.136	0.111	0.017	−0.121
Triglyceride (mg/dL)				1	**0.243***	0.120	0.064	**−0.353**†	−0.010	**0.368**†	0.120	0.174	−0.141	−0.242
HOMA index					1	**0.622**†	−0.021	−0.067	−0.175	**0.254***	0.114	0.014	0.014	−0.032
Glucose (mg/dL)						1	0.071	0.074	**−0.227***	0.145	0.174	0.012	0.025	0.085
HbA1C (%)							1	0.093	−0.088	0.003	−0.025	0.145	−0.088	0.121
HDL cholesterol (mg/dL)								1	0.117	**−0.291**†	−0.142	**−0.201***	**0.215***	0.287
LDL cholesterol (mg/dL)									1	−0.130	0.107	0.244	0.014	−0.024
BMI percentile										1	0.105	−0.130	0.127	0.022
Leptin (ng/mL)											1	0.170	0.200	−0.121
Omentin-1 (pg/mL)												1	−0.088	−0.136
Vaspin (pg/mL)													1	−0.047
Adiponectin (μg/mL)														1

BMI, body mass index; ALT, alanine aminotransferase; AST, aspartate aminotransferase; LDL, low density lipoprotein; HDL, high density lipoprotein.
*Correlation is significant at the 0.05 level (2-tailed); †Correlation is significant at the 0.01 level (2-tailed).

Table 3. Comparison of laboratory parameters according to the degree of hepatosteatosis

Variables	NAFLD Grade-1 hepatosteatosis (n = 19)	NAFLD Grade-2 hepatosteatosis (n = 12)	NAFLD Grade-3 hepatosteatosis (n = 1)	p value
Age (yr)	11.50	14.00	15.00	0.507
AST (U/L)	13.95	18.25	32.05	0.375
ALT (U/L)	17.73	22.00	24.98	0.579
Total cholesterol (mg/dL)	129.10	141.80	163.80	0.189
Triglyceride (mg/dL)	72.05	111.00	141.10	0.744
HOMA index	2.50	4.00	7.00	0.147
Glucose (mg/dL)	89.93	93.65	98.45	0.767
HBA1C (%)	5.21	5.39	5.57	**0.036***
HDL cholesterol (mg/dL)	33.85	40.75	47.83	0.933
LDL cholesterol (mg/dL)	62.52	79.59	92.88	0.154
Leptin (ng/mL)	12.47	15.96	18.54	0.503
Omentin-1 (pg/mL)	220.83	241.82	262.82	0.502
Vaspin (pg/mL)	1,095.50	1,204.38	1,312.51	0.798
Adiponectin (μg/mL)	11.42	12.84	14.82	0.868

NAFLD, non-alcoholic fatty liver disease; AST, aspartate aminotransferase; ALT, alanine aminotransferase; HDL, high density lipoprotein; LDL, low density lipoprotein.
*Kruskal Walls test, bold indicates statistically significant ($p < 0.05$).

While we compared the leptin, omentin-1, vaspin and adiponectin levels of non-obese children with those of the O/O group with hepatosteotosis (grade 1, 2–3), it was observed that as the degree of hepatosteotosis increased, leptin ($p = 0.004$) and omentin-1 ($p = 0.001$) levels were increased. There was no significant change in vaspin level ($p = 0.128$) (**Table 4**).

Table 4. Comparison of adipokine levels of the obese/overweight group with hepatosteatosis and non-obese children

Adipokine level (mean ± SD)	Controls (n = 32)	NAFLD Grade 1 (n = 19)	NAFLD Grade 2–3 (n = 13)	p value
Leptin (ng/mL)	12.49 ± 2.92	15.61 ± 3.30	16.40 ± 2.67	**0.004***
Omentin-1 (pg/mL)	197.49 ± 37.45	250.38 ± 43.28	251 ± 35.42	**0.001***
Vaspin (pg/mL)	1,355.30 ± 280.90	1,190.67 ± 178.03	1,201 ± 162.25	0.128
Adiponectin (μg/mL)	10.44 ± 2.05	13.92 ± 2.86	12.76 ± 3.11	**0.001***

Since there was only one patient in grade 3, values in grade 3 has been combined with those in grade 2. NAFLD was defined in 3 grades according to USG findings: Grades 1, 2 and 3. According to USG findings, only mild diffuse echo increase in liver was grade 1; mild effacement of intrahepatic vascular appearance with moderate echo increase in liver grade 2; Significant effacement in the intrahepatic vascular structures with advanced echo increase in the liver and detection of effacement in the posterior view of the right lobe were evaluated as grade 3 hepatosteatosis.
NAFLD, non-alcoholic fatty liver disease.
*Kruskal Walls test, bold indicates statistically significant ($p < 0.05$).

https://doi.org/10.7762/cnr.2022.11.3.194

Table 5. Comparison of adipokine levels in obese/overweight children with and without hepatosteatosis

Adipokin level (mean ± SD)	Without hepatosteatosis (n = 17)	With hepatosteatosis (n = 32)	p value*
Leptin (ng/mL)	15.26 ± 3.27	15.65 ± 3.09	0.721
Omentin-1 (pg/mL)	243.85 ± 18.49	244.97 ± 39.65	0.825
Vaspin (pg/mL)	1,202.80 ± 137.58	1,204.53 ± 166.64	0.933
Adiponectin (μg/mL)	12.93 ± 2.24	13.78 ± 2.84	0.433

*Student's t-test.

We divided O/O children into two groups with and without hepatosteatosis and compared the adipokine levels. There was no significant difference observed (**Table 5**).

DISCUSSION

Obesity is an increasing health problem that has become an epidemic today. Adipose tissue is a source of inflammation and is considered as a complex and active metabolic endocrine organ that produces various cytokines. Adipose tissue-derived adipokines play roles in the regulation of many processes such as inflammation, diabetes, energy metabolism and atherosclerosis [15].

We aimed to examine the adipokines (omentin, vaspin, adiponectin and leptin) level in O/O children and to evaluate correlation with other laboratory parameters and hepatosteatosis. Vaspin, omentin-1, adiponectin, and leptin have been identified as novel adipokines and have been thought to be related with components of hyperinsulinemia, inflammation and metabolic syndrome [16].

Some studies have been conducted to show the relationship between vaspin and obesity and different results were obtained. Auguet et al. [16] reported that vaspin mRNA expression was significantly higher in visceral adipose and subcutaneous tissue without increasing serum vaspin levels. Sperling et al. [17] found no difference in vaspin concentrations in obese individuals divided into two different groups with normal and abnormal results in glucose tolerance test. Klöting et al. [18] revealed body fat percentage as the strongest predictor of visceral vaspin and insulin sensitivity as the strongest predictor of subcutaneous vaspin mRNA expression. In addition, Youn et al. [19] found vaspin concentration to be higher in women and showed that high serum vaspin levels were associated with impaired insulin sensitivity and obesity. Chang et al. [20], conducted a study to analyse the relationship between abdominal adiposity and serum vaspin concentrations, that showed a strong correlation between serum vaspin level and visceral adipose tissue depot area (VAT) when HOMA-IR was high.

In current study, vaspin levels were found to be significantly lower in the O/O group (1,203.94 ± 155.71 pg/mL) compared to the non-obese group (1,355.30 ± 280.90 pg/mL) ($p = 0.008$). There was no significant correlation between vaspin and insulin resistance (HOMA-IR). We found that vaspin had a significant positive correlation with HDL-cholesterol in the O/O group ($p < 0.01$). Also there was no significant difference in the comparison of vaspin levels between genders. We could not analyze vaspin expression in adipose tissue.

In some studies conducted in recent years, it has been reported that leptin and adiponectin levels were associated with insulin resistance. Pîrsean et al. [21], showed that despite normal

https://doi.org/10.7762/cnr.2022.11.3.194

serum lipid and glucose in obese children, salivary leptin level was found to be significantly higher. Again, Bodini et al. [22] showed that leptin is measurable in exhaled breath in children, and exhaled breath-leptin levels were significantly higher in obese individuals and asthmatics compared to healthy subjects. In our study, leptin and adiponectin levels were found to be significantly higher in the O/O group compared to the controls ($p < 0.01$).

Taniguchi et al. [23] showed a positive correlation between HOMA-IR and leptin and a negative correlation between adiponectin and HOMA-IR in non-obese Type 2 DM patients. No correlation was found between HOMA-IR and adipokines in this study. Although not significant, we found a positive correlation between leptin and HOMA-IR in the O/O group, and a negative correlation between the HOMA-index and adiponectin.

Silha et al. [24] reported that adiponectin levels were significantly lower in obese compared to lean subjects and higher in females, but they did not show a significant correlation with the HOMA index. Leptin levels were higher in obese women and correlated with the HOMA index and resistin. In the study of Gulturk et al. [25], leptin levels in women were also found to be significantly higher than in men in both T2DM, and healthy participants. In our study, there was no significant difference between male and female in leptin and adiponectin levels in the O/O group.

Many studies in adults have suggested that omentin-1 is inversely correlated to obesity and insulin resistance. Auguet et al. [16] revealed that serum omentin-1 levels were significantly lower in the morbidly obese subjects and inversely correlated with the metabolic syndrome (MS). The expression of omentin-1 in visceral adipose tissue was significantly lower in morbidly obese women compared to controls. Çatoi et al. [26] found in their study that omentin-1 levels were decreased in morbidly obese compared to normal-weight healthy individuals and were inversely related to chronic inflammation and dyslipidemia. Rothermel et al. [27] stated in their study that omentin-1 concentrations were significantly lower in obese children compared to children with normal weight and negatively associated with HOMA-IR. It was observed that the serum level of omentin-1 increased with weight loss.

In our study, omentin-1 levels were found to be significantly higher in the O/O group (244.59 ± 33.61 pg/mL) compared to the control group (197.49 ± 37.45 pg/mL) ($p = 0.001$). We also found a significant ($p < 0.01$), negative correlation between omentin-1 and HDL-cholesterol in the O/O group. Omentin-1 was not correlated with other laboratory data. There was no difference on comparing gender.

We compared the levels of vaspin, omentin-1, leptin and adiponectin in non-obese and O/O children with hepatosteatosis and found that leptin, omentin-1 and adiponectin levels were significantly higher in the hepatosteatosis group. There was no significant difference in vaspin levels. Our data support the concept that elevated adipokine levels are closely linked with hepatosteotosis. Also we showed that as the degree of hepatosteatosis increased in children with NAFLD, serum leptin and omentin-1 levels increased. Elevated leptin and omentin-1 levels may be appropriate serum markers to predict advanced hepatosteotosis in children with NAFLD.

Polyzos et al. [28] stated in their meta-analysis studies that leptin levels are extremely high in individuals with hepatosteatosis compared to the control group and higher levels of circulating leptin were associated with increased severity of hepatosteatosis.

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In another meta-analysis study by Polyzos et al. [29], it was found that adiponectin levels were lower in individuals with hepatosteatosis compared to the control group. In the study of Lebensztejn et al. [30], serum leptin level was higher and adiponectin was lower in obese children with NAFLD compared to controls. Adiponectin was correlated with HOMA-IR and ultrasonographic grades of liver were negatively correlated with adiponectin in the same study. Boyraz et al. [31] showed that leptin was higher in obese children with NAFLD and adiponectin was negatively correlated with ALT and ultrasonographic grading. They suggested that adiponectin and leptin were the markers for predicting metabolic syndrome and NAFLD. Abenavoli et al. [32] found similar results and they suggested that leptin level was higher; adiponectin was lower in overweight patients with NAFLD. Also they revealed that serum adiponectin and leptin levels might be used as diagnostic markers to determine the presence of NAFLD in overweight patients.

Yılmaz et al. [33] found that omentin-1 levels were high in individuals with biopsy-proven NAFLD disease and serum omentin-1 levels were significantly associated with C-reactive protein and the degree of hepatocyte ballooning.

Aktas et al. [34] stated in their study that the vaspin level was high in individuals with hepatosteatosis and serum vaspin levels showed a statistically significant association with CRP and liver fibrosis scores. There are studies in literature that, vaspin levels observed higher in the patients with histologically confirmed NAFLD [35], and others reported similar vaspin levels in NAFLD patients and control [36].

Study limitations

The expression of adipokine levels in visceral adipose tissue couldn't be analysed and hepatosteatosis could not be proven with liver biopsy in the obese children.

In conclusion, while serum leptin, omentin-1 and adiponectin levels were higher, vaspin levels were significantly lower in the O/O group. Furthermore, omentin-1, leptin and adiponectin levels were significantly higher in the obese children with hepatosteatosis group. The cut-off value of adipokine levels related to the degree of hepatosteatosis can be shown in future larger studies. The reason why some values are contrary to the literature may be due to the presence of overweight subjects in current study.

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