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Research Article

EVALUATION OF THE RELATIONSHIP BETWEEN PLATELET INDICES AND LIPID PROFILE IN OBESE ADOLESCENTS WITH HEPATOSTEATOSIS

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ABSTRACT

Introduction: Obesity leads to an increased risk of cardiovascular disease both as an independent factor and by causing metabolic disorders such as hepatosteatosis, hypertension, dyslipidemia, and insulin resistance. Changes in platelet indices have a significant impact in thrombotic and pre-thrombotic events. In our study, we aimed to evaluate platelet indices such as the platelet count (PLT), mean platelet volume (MPV) and platelet distribution width (PDW) that have a role in the atherothrombotic process and to investigate their relationship with lipid profile tests such as total cholesterol, triglycerides, HDL-cholesterol and LDL-cholesterol in obese adolescents with hepatosteatosis.

Methods: We gathered data by retrospectively investigating the files of 68 patients aged 10-16. Adolescents with a body mass index (BMI) at or above the 95th percentile according to age and gender were described as obese.Patients were split into three groups based on USG results as following: obese adolescents with hepatosteatosis, obese adolescents without hepatosteatosis, and control group of healthy adolescents. The groups were compared in terms of laboratory tests, gender, age, and BMI. The data were statistically evaluated usingSPSS software package.

Results: PDW values of the obese group with hepatosteatosis were statistically significantly lower than those of the healthy control group ($p=0.017$; $p<0.05$). There was no statistically significant difference between the 3 groups in terms ofPLT and MPV levels ($p>0.05$). There was a significant positive correlation betweenPLT and total cholesterol, and LDL-cholesterol ($p<0.05$).

Conclusion: Obese adolescents with hepatosteatosis had lower PDW levels. On the other hand, PLT and MPV levels had no relationship with obesity and hepatosteatosis in adolescents. Moreover, the positive correlation betweenPLT, total cholesterol, and LDL-cholesterol indicates that platelets and lipids might have a cooperative role inatherothrombotic complications in obese adolescents with hepatosteatosis.

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INTRODUCTION

Nonalcoholic fatty liver disease (NAFLD) is described as the accumulation of fat, especially triglycerides, in the liver in a higher proportion than 5% of the liver's weight, and histopathologically, with more than 5% of the hepatocytes being filled with fat vacuoles [1, 2]. Fatty liver affects 22-52% of obese children and 10-25% of adolescents [3]. Hepatosteatosis manifests as a complication of obesity. Obesity, which is characterized by the excessive accumulation of adipose tissue and lipids that cause ectopic lipid accumulation in different tissues, is a chronic proinflammatory condition that exhibits increased cytokine levels [4]. Obesity increases the risk of cardiovascular morbidity and mortality through various ways. These include an increased dyslipidemia, prothrombotic tendency, type 2 diabetes mellitus, and hypertension associated with obesity [5].

Obese adolescents can also exhibit changes in lipid profile tests such as an increased serum total cholesterol, triglycerides, and low density lipoprotein (LDL- cholesterol) levels and decreased high density lipoprotein (HDL-cholesterol) levels [6]. The most common cause of cardiovascular diseases is atherosclerosis, which is accompanied by thrombosis [7]. Platelets play an important role in the pathophysiology of atherothrombotic disease [8]. Changes in platelet parameters are

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of great importance in thrombotic and pre-thrombotic events [9]. Platelet count (PLT), mean platelet volume (MPV), and platelet distribution width (PDW), which indicate the percentage of change in platelet size, are platelet indices that are associated with platelet activity [10].

In our study, we aimed to evaluate PLT, MPV, and PDW levels that have a role in the atherothrombotic process and to investigate their relationship with the lipid profile of obese adolescents with hepatosteatosis.

Experimental Section

In our study, 27 non-obese healthy adolescents and 41 obese adolescents who presented at the pediatric outpatient clinic of our hospital between July and December 2018 constituted the healthy control and obese patient groups, respectively. We obtained the data for our study by retrospectively investigating the files of 68 patients aged between 10 and 16. Body mass index (BMI) was obtained by dividing the weight of the patient in kg by the square of the height in meters. Adolescents with a (BMI) at or above the 95th percentile according to age and gender were described as "obese" individuals [11]. A total of 20 patients with grade 2-3 hepatic steatosis according to the abdominal ultrasonography (USG) results constituted the group of "obese adolescents with hepatosteatosis", 21 obese patients without hepatosteatosis constituted the group of "obese adolescents without hepatosteatosis" and 27 patients without hepatosteatosis according to abdominal USG and with a BMI at the normal percentile, according to age and gender, constituted the group of "healthy control adolescents". Laboratory tests [HbA1c, glucose, urea, creatinine, aspartate amino transferase (AST), alanine amino transferase (ALT), total cholesterol, triglycerides, HDL-cholesterol, LDL-cholesterol, hemoglobin (Hb), hematocrit (Hct), red blood cell (RBC), white blood cell (WBC), platelet indices, i.e. PLT, MPV, and PDW], gender, age, and BMI were compared between the groups. Correlations between platelet indices and other parameters were investigated. LDL-cholesterol level was calculated utilizing the Friedewald formula [LDL-cholesterol= total cholesterol - (HDL-cholesterol) - (triglyceride/5)]. The data were statistically evaluated using the SPSS software package.

The subjects who were under a treatment that could affect the platelet functions (aspirin, warfarin, heparin etc.), those who smoked and consumed alcohol, those with a chronic disease, infection, metabolic and endocrine disease, those receiving drug therapy such as corticosteroids, and patients who had a malignancy were excluded from the study.

Blood samples were placed in ethylenediamine-tetraacetic acid (EDTA) tubes and PLT, MPV, and PDW levels were determined with a routine hemogramautoanalyzer (Mindray brand, BC6800 model, 2017, China). HbA1c test was carried out in an autoanalyzer (Biorad, Variant II turbo, 2010, Japan) using a high-performance liquid chromatography method while glucose, urea, creatinine, AST, ALT, total cholesterol, triglycerides, HDL-cholesterol, and LDL-cholesterol levels were measured employing an autoanalyzer (Beckman Coulter, AU 5800 model, 2013, USA) using the colorimetric method.

Ethics committee approval: The approval for this study was obtained from the Ethics Committee of the University of Health Sciences Istanbul Okmeydanı Health Practice and Research Center.

Statistical Analysis: IBM SPSS Statistics 22 (IBM SPSS, Turkey) software was used. The normal distribution of the parameters was analyzed using the Shapiro Wilk test. In addition to using descriptive statistics (mean, standard deviation, frequency), for the comparison of the quantitative data of parameters with a normal distribution, One Way Anova test was used in intergroup comparisons as well as Tukey's HSD test and Tamhane's T2 test in determining the group that causes the change. For non-normally distributed parameters, Kruskal Wallis test was used in intergroup comparisons and Mann Whitney U test in determining the group that causes the difference. Student-t test was utilized in comparing normally distributed parameters between two groups, and Mann Whitney U test in comparing the non-normally distributed parameters between two groups. Continuity (Yates) Correction was used in the comparison of qualitative data. Pearson correlation analysis was applied to analyze the correlations between normally distributed parameters. The level of significance was accepted as $p<0.05$.

RESULT AND DISCUSSION

Our study consisted of 68 adolescents in total, i.e. 23 (33.8%) females and 45 (66.2%) males. The mean age of the subjects was 13.03±1.98. BMI values ranged from 16.76 to 43.06. There was no statistically significant difference between the groups in terms of age, gender, HbA1c, glucose, urea, creatinine, AST, total cholesterol, triglycerides, HDL-cholesterol, LDL-cholesterol, WBC, RBC, Hb, Hct, PLT, and MPV ($p > 0.05$).

There was a statistically significant difference between the groups in terms of BMI, ALT, and PDW values ($p < 0.05$).
BMI and ALT values of the obese adolescents group with hepatosteatosis were statistically significantly higher than the same of the other groups ($p < 0.001$), whereas the BMI and ALT values of the obese adolescents group without hepatosteatosis were statistically significantly higher when compared to the healthy control group ($p < 0.001$ and $p = 0.047$, respectively).

PDW values of the obese adolescent group with hepatosteatosis were statistically significantly lower than those of the healthy control group ($p = 0.017$; $p < 0.05$). There was no statistically significant difference between the other groups in terms of PDW values ($p >0.05$) (Table 1).

Table 1 Data of the obese group with and without hepatosteatosis and healthy control group

	Obese without hepatosteatosis (n:21)	Obese with hepatosteatosis (n:20)	Healthy Control group (n:27)	P
	Mean±SD	Mean±SD	Mean±SD	
Age	12.43±1.94	13.55±1.88	13.11±2.04	[1]0.189
Gender *n (%)*				
Female	7 (33.3%)	6 (30%)	10 (37%)	[2]0.879
Male	14 (66.7%)	14 (70%)	17 (63%)	
BMI	29.54±2.62	34.14±4.5	23.69±3.5	[1]<0.001*
HbA1c (%)	5.37±0.27	5.46±0.38	5.21±0.38	[1]0.058
Glucose (mg/dL)	86.62±9.32	89.3±8.27	87.41±7.92	[1]0.583
Urea (mg/dL)	25.33±6.21	25.9±6.65	24.33±5.74	[1]0.677
Creatinine (mg/dL)	0.53±0.1	0.55±0.14	0.54±0.15	[1]0.840
AST *(median)* (U/L)	22.9±4.64 (22)	25.75±7.55 (25.5)	21.44±5.42 (22)	[3]0.137
ALT *(median)* (U/L)	19.57±7.82 (17)	28.45±10.7 (26.5)	15.81±6.25 (14)	[3]<0.001*
Total Cholesterol (mg/dL)	161.67±22.17	165.35±38.36	152.33±33.25	[1]0.355
Triglyceride *(median)* (mg/dL)	108.29±51.81 (96)	110.65±55.73 (91)	81.74±39.83 (69)	[3]0.077

HDL-cholesterol *(median)* (mg/dL)	46.38±9.05 (48)	44.25±11.16 (40.5)	48.63±11.57 (47)	[3]0.295
LDL-cholesterol (mg/dL)	92.86±19.49	103.75±27.96	87.26±24.78	[1]0.077
WBC (x10^3/µL)	7.77±1.33	8.14±1.49	7.79±1.81	[1]0.695
RBC *(median)* (x10^6/µL)	4.92±0.37 (4.8)	5.01±0.33 (4.9)	5.08±0.49 (5.1)	[3]0.556
Hb (g/L)	131.29±11.19	133.65±9.99	138.63±14.44	[1]0.113
Hct (%)	39.49±2.85	40.27±2.96	41.27±3.99	[1]0.195
PLT (x10^3/µL)	297.67±42.18	324.15±75.12	295.52±84.52	[1]0.347
MPV (fL)	9.45±0.72	9.09±1.03	9.49±0.94	[1]0.293
PDW (%)	15.85±0.22	15.73±0.35	16.03±0.35	[1]0.007*

[1]One Way Anova Test,
[2]Continuity (Yates) Correction,
[3]Kruskal Wallis Test *p<0.05

There was a statistically significant positive correlation between the PLT and total cholesterol, LDL-cholesterol (p <0.05) (Table 2).

Table 2 Correlations between platelet indices and other parameters in the obese adolescent group with hepatosteatosis

		BMI	HbA1c	Total Cholesterol	Triglyceride	HDL-cholesterol	LDL-cholesterol	WBC	RBC	Hb	Hct
PLT	r	0.089	0.025	0.554	0.369	0.285	0.512	0.104	-0.075	-0.266	-0.224
	P	0.709	0.917	0.011*	0.109	0.223	0.021*	0.664	0.753	0.257	0.342
MPV	R	0.246	0.226	-0.371	-0.066	-0.353	-0.312	-0.006	0.011	0.079	0.085
	P	0.295	0.338	0.107	0.782	0.127	0.181	0.981	0.962	0.742	0.722
PDW	R	0.120	0.080	-0.246	0.011	-0.225	-0.201	-0.232	0.366	0.435	0.426
	P	0.613	0.736	0.296	0.962	0.339	0.395	0.325	0.112	0.055	0.061

Pearson correlation analysis: **p<0.05*

Cardiovascular diseases were discovered to be associated with an increased MPV and elevated platelet levels [(12)]. In addition, it was reported in studies that MPV could be used as a marker in monitoring early atherosclerosis, metabolic syndrome, and nonalcoholic steatohepatitis, since it is correlated with platelet functions and activation [(13)]. In obesity, a low-grade inflammatory process is observed with the increase in platelet count and incidence of thrombosis [(14)]. According to a study by Özsu and Yazıcıoğlu, there was no significant variation in the PLT, MPV, and PDW values between the obese children group and control group [(15)]. Emeksiz found that there was no difference between the control group and obese group in terms of PLT, MPV and PDW values [(16)]. Similar to these results, we found that there was no significant difference between the obese adolescents without hepatosteatosisand the control group in terms of the platelet indices, i.e. PLT, MPV, and PDW levels. However, Çoban et al. reported that obese subjects had an increased MPV, and this was a risk factor for cardiovascular disease [(17)].

The foundations of atherosclerosis are laid in childhood. Early lesions of this process referred to as fatty streaks are seen in the intima of the aorta at the age of 3 and in coronary arteries at the age of 14 [(18)]. The subclinical inflammatory process plays a role in the pathogenesis of obesity-associated hepatosteatosis and atherosclerosis [(19)]. As is the case with obesity, NAFLD is also associated with atherosclerosis and cardiovascular disease [(20)].
Arslan and Makay reported increased MPV values in the presence of risk factors such as obesity and NAFLD in an adolescent population [(20)].Woul-young et al. mentioned the relationship between NAFLD and increased MPV in their study conducted on obese subjects [(21)]. Kertmen et al. compared the PLT and MPV values between two groups consisting of children aged between 4 and 16 with and without hepatosteatosis and did not find any significant difference between the groups [(22)]. In our study, we did not find a significant difference in PLT and MPV values of obese adolescents with hepatosteatosis. However, we found that obese adolescents with hepatosteatosishad significantly decreased PDW valueswhen compared to the control group and obese adolescents without hepatosteatosis group. We could not directly compare this result with the literature because our study is the first one that investigates PDW values in obese adolescents with hepatosteatosis.

Some studies in the literature mention decreased PDW and MPV values. Decreased MPV values were observed in rheumatoid arthritis and ulcerative colitis [(23, 24)]. It was mentioned that a decreased MPV is caused by the increased consumption of large platelets in inflammatory conditions [(25)].

Since megakaryocyte maturation, platelet production, and platelet size can be modulated by cytokines such as interleukin-3 (IL-3), interleukin-6 (IL-6), granulocyte colony stimulating factor, and macrophage colony stimulating factor, varying levels of biomolecules that play a role in inflammation can have an effect on platelet size. Furthermore, the dysregulation of bone marrow cells (including megakaryocytes) can contribute to the decrease in MPV and PDW [(26)].
In the light of this information, it is considered that PDW values are decreased in correlation with the inflammation levels specific for the group of obese adolescents with hepatosteatosis, and PLT and MPV levels do not exhibit any change. Contradictory results were obtained in both obese patients and subjects with hepatosteatosis, especially in terms of the MPV values. As a matter of fact, we observed that obese patients with hepatosteatosis had increased platelet levels, although not statistically significant. This might have reduced PDW values.However, further studies are needed on this subject.

Lipids play a role in the pathogenesis of cardiovascular diseases. A decreased HDL level is a potent and independent risk factor for cardiovascular diseases and atherosclerosis [(27)]. HDL-cholesterol reduces the oxidation of LDL-cholesterol, leads to a decreased platelet activation, and reduces the expression of adhesion molecules, thereby regulating platelet functions and exerting an antiplatelet effect [(28)]. Moreover, it was reported that an increased MPV could affect the development or progression of atherosclerotic activity in familial hypercholesterolemia, although its precise biological pathways could not be entirely described [(29)]. These data indicate that there is a relationship between platelet indices and the lipid profile, both playing a role in the atherothrombotic process.

Varol et al. [(30)] reported increased MPV values in subjects with low HDL-cholesterol levels [(30)]. Woul-young et al. [(21)] found a negative correlation between MPV and HDL-cholesterol and a

positive correlation between MPV and triglyceride level but did not report any correlations between MPV and total cholesterol, and LDL-cholesterol in the obese group with hepatosteatosis[21]. Karakılçık et al. did not find any correlations between PLT, MPV and lipid profile tests, but reported a negative correlation between PDW and total cholesterol, HDL-cholesterol, and LDL-cholesterol in a group of 22 young males [31]. According to the correlation analyses in our study, there was no correlation between MPV, PDW, and the lipid profile, on the other hand there was a positive correlation between PLT, total cholesterol, and LDL-cholesterol in obese adolescents with hepatosteatosis. This implies that lipids and platelets, which have a role in the atherothrombotic process, could also have a cooperative role in cardiovascular complications seen in obese adolescents with hepatosteatosis.

Emeksiz et al. found that obese subjects had elevated triglyceride and ALT levels and low HDL-cholesterol, but no change in total cholesterol and LDL-cholesterol levels [16]. Kertmen et al. reported increased BMI and ALT levels in the presence of hepatosteatosis[22]. In parallel with these data, we found BMI and ALT values consistent with the literature, but determined no significant difference in lipid profile tests in obese adolescents with hepatosteatosis.

Our study had some limitations. First of all, our study populations were limited and the patients did not have any cardiovascular complications yet. Therefore, we should validate our results with larger groups of adolescent subjects who also have cardiovascular complications.

Consequently, we think that platelets and lipids might have a combined roleand they should be evaluated together in terms of possible atherothrombotic complications. We believe that more comprehensive studies would be useful for clarifying the relationships between the lipid profile and platelets.

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