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Diet induced weight loss reduces mean platelet volume in people with obesity

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Abstract

Objectives: To investigate is there a relationship between obesity parameters, mean platelet volume (MPV) and neutrophil lymphocyte ratio (NLR), and to examine is there a change in MPV and NLR level with weight loss.

Material and Methods: The retrospective study consisted of two groups as the group losing weight after 24 weeks of diet (WLG) and the control group not losing weight (CG). Body mass index (BMI), MPV, NLR and lipid values of each group were recorded at the beginning and at the end of the study.

Results: The study consisted of 186 individuals 94 of whom were in WLG and 92 were in CG. There was a statistically significant positive correlation between the initial value of MPV and BMI in WLG ($p < 0.05$). There was a significant difference existed in the initial and final lipid parameters of WLG ($p<0.0001$). Changes in initial and final MPV values in WLG and CG were different from each other ($p < 0.01$). The difference between the initial and final MPV values and BMI values in WLG was statistically significant ($P < 0.001$). No significant correlation was found between obesity and weight loss with NLR.

Conclusions: There was a statistically significant correlation between MPV and BMI. MPV levels decreased in obese individuals with weight loss. No correlation existed between BMI and NLR.

Keywords: Diet, weight loss, mean platelet volume, neutrophil lymphocyte ratio, obesity, body mass index

Introduction

The importance of inflammatory markers is increasing as coronary artery diseases are currently the most common causes of death in the world, which is related to inflammation (1). Therefore, a number of studies have recently been conducted on inflammation and biomarkers which reflect inflammatory conditions (1, 2). In obesity whose prevalence is gradually increasing due to the decrease in physical activity and increase in high-energy food consumption, body mass index (BMI) above normal increases mortality and morbidity (3, 4). According to World Health Organization (WHO), an individual is defined as overweight (preobese) if BMI is above 25 kg / m² and as obese if BMI is above 30 kg / m² (4). Furthermore, obesity plays an important role in the formation of many pathological conditions. The most frequent conditions can be listed as cardiovascular disease, cerebrovascular disease, hypertension (HT), diabetes mellitus (DM), dyslipidemia, infertility, certain types of cancer such as prostate, breast and colon (5).

Although obesity-related causes of inflammation are not fully understood, it is estimated that it occurs due to direct activation of immune cells in the circulatory system (6, 7).

The inflammatory process also occurs in liver, pancreas, adipose and muscle tissue with circulation. Adipose tissue has both initiatory and contributory role in systemic inflammation (6, 7).

Mean platelet volume (MPV) associated with cardiovascular and cerebrovascular diseases and accepted as an indication of atherosclerosis is a parameter used to evaluate the size and activity of platelets (8). The volume of activated platelets increases, which causes MPV rise. Large platelets including more granules and produces more vasoactive and prothrombotic factors make more aggregation compared to small platelets (9, 10). MPV is known to increase in acute myocardial infarction, acute ischemic stroke, preeclampsia and renal artery stenosis (8). Additionally, in some studies, it has been discovered that MPV is positively correlated with

BMI and high in obesity (11). In recent studies on humans and animals, it has been demonstrated that the change in neutrophil and lymphocyte numbers poses a potential risk for the development of obesity-related metabolic disorders (12, 13).

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It is believed that Neutrophil / Lymphocyte ratio (NLR) is a marker of systemic inflammation and has a prognostic significance for cardiovascular diseases. These hematological markers are important in terms of making assessment without creating extra costs during the complete blood count (14). Although there are a number of studies conducted on the relationship between MPV and obesity and no common conclusion is drawn. In this study, our aim is to investigate the relationship between obesity parameters, which are known to accelerate the development of atherothrombosis and the incidence of cardiovascular mortality and morbidity, MPV and NLR. We also aim to examine whether there is a change in MPV and NLR levels with weight loss thanks to a diet plan in obese individuals.

Material and Methods

This study was conducted retrospectively after the approval of Ethics Committee of Balıkesir University School of Medicine was granted. When individuals go to Balıkesir University School of Medicine Health Application and Research Hospital with complaints related to being overweight, the arterial blood pressure is measured and physical examination is performed. After those procedures, complete blood count and biochemical tests (12 hour fasting glucose, total cholesterol, LDL cholesterol, HDL cholesterol, triglycerides, and HOMA-IR TSH) are performed routinely. For complete blood count, blood sample is taken from the antecubital vein, put into tubes with EDTA and studied with Beckman Coulter LH 780 Hematology Analyzer (Beckman Coulter, Inc., CA, USA) device within an hour. If metabolic syndrome (MS), DM, and dyslipidemia are detected in those individuals medical treatment is given and they are directed to diet clinic. Measuring weight and height, BMI of obese individuals are calculated through weight (kg) / (height) ^{2} (m^2) formulas, and appropriate nutrition programs are given by dieticians. Weight changes of these individuals are followed for 12-week periods. During the controls in internal medicine clinic, detailed physical examination, complete blood count and biochemical tests are performed, and individuals meet the dieticians again. We scanned the files of obese individuals who were directed to dieticians between 2013-2015, had $\geqslant$ 30 kg / m2 body mass index (BMI) according to WHO classification, and followed at least 24 weeks of dieting. The individuals who were found to have DM (fasting blood glucose $\geqslant$126 and hemoglobin A1c> 6.4%), cardiovascular disease, HT (systolic blood pressure $\geqslant$140 mm Hg diastolic blood pressure $\geqslant$90), cerebrovascular disease, liver failure, renal failure, smoking and alcohol habit were excluded from the study. On the other hand, individuals who were eligible for the study were divided into two groups as weight loss group (WLG) including individuals with body weight change higher than 5%, and the control group (CG) consisting of individuals who had weight loss less than 5%. The initial values of WLG and CG were recorded as MPV1, NLR1, BMI1 and the values after 24 weeks were recorded as MPV2, NLR2 and BMI2.

Diet Plan

Patients' daily energy requirements were calculated according to age, gender, weight and height. A weight loss diet was implemented ensuring that energy intake was above the basal metabolic rate (BMR). Gerrior et al. formulae was used to calculate BMR: "BMR = 247 – 2.67 × age (year) + 401,5 × height boy (meter) + 8,6 × weight (kg)" (15). The diet consisted of 55-60% carbohydrates 12-15 % protein and 25-30 % lipid and the energy value of the diets given ranged between1300-1800 kcal.

Statistical analysis: The values of the obese group losing weight as a result of dieting and control group losing weight without diet were evaluated using SPSS software version 20.0 (SPSS Inc, Chicago, IL, USA). Chi square test was used for determining the relations of gender and age between groups. In addition, groups were compared using the Student's t-test. The groups' initial and final results were compared using the Paired sample t test. The correlation between BMI, MPV and NLR was calculated via Pearson correlation test. MPV changes within the group were demonstrated through univariate covariance analysis. $P<0.05$ value was accepted as significant.

Results

186 obese individuals (153 females, 33 males) appropriate for the study criteria were included in the study. WLG consisted of 94 individuals (79 females and 15 males) between 21-64 years old, while CG included 92 individuals (74 females and 18 males) between 19-64 years old. No difference was found between groups in terms of gender and age distribution ($P>0.05$). Moreover, no significant difference existed between MPV values of males and females ($P>0.05$). The mean MPV1 level of all individuals in the study was found to be 8.38 ± 0.95 while the mean MPV2 was 8.21 ± 0.94. The mean MPV1 of WLG was 8.40 ± 0.95 whereas the mean MPV2 was found to be 7.93 ± 0.84 (Table 1). When the initial BMI values of both groups were compared, a significant difference was found ($p<0.05$). The mean BMI 1 value of WLG was 36.68 ± 5.72, whereas it was 34.62 ± 5.09 for CG. At the end of the study, when the mean BMI 2 of WLG (32.17 ± 5.04) and the

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Table 1: The anthropometric measurements and haematological values of non-weight loss and weight loss groups

	Non weight loss (n: 97)			Weight loss (n: 101)		
	First count	Last count	p	First count	Last count	p
Body weight (kg)	89.0 ± 14.2	90.5 ± 14.5	0.001	94.3 ± 15.3	82.8 ± 14.0	0.001
BMI (kg/m2)	34.6 ± 5.0	35.2 ± 5.1	0.001	36.6 ± 5.7	32.1 ± 5.0	0.001
Total cholesterol (mg/dL)	224.1 ± 46.7	217.7 ± 43.2	0.137	239.1 ± 55.0	190.1 ±39.8	0.001
LDL- cholesterol (mg/dL)	138.9 ± 42.2	139.7 ± 36.5	0.161	152. 5± 52.5	107.4 ± 33.9	0.001
HDL- cholesterol (mg/dL)	50.2 ± 12.1	49.6 ± 11.2	0.462	50.8 ± 11.6	56.4 ± 10.4	0.001
Triglyceride (mg/dL)	175.1 ± 65.6	170.7 ± 65.6	0.359	190.3 ± 85.8	134.0 ± 55.1	0.001
MPV (fl)	8.3 ± 0.9	8.5 ± 0.9	0.023	8.4 ± 0.9	7.9 ± 0.8	0.001
NLR (%)	1.6 ± 0.9	1.7 ± 1.2	0.405	1.8 ± 0.9	1.9 ± 0.8	0.001

BMI: body mass index; LDL: Low Density Lipoprotein; HDL: High Density Lipoprotein;
MPV: mean platelet volume; NLR: neutrophil lymphocyte ratio

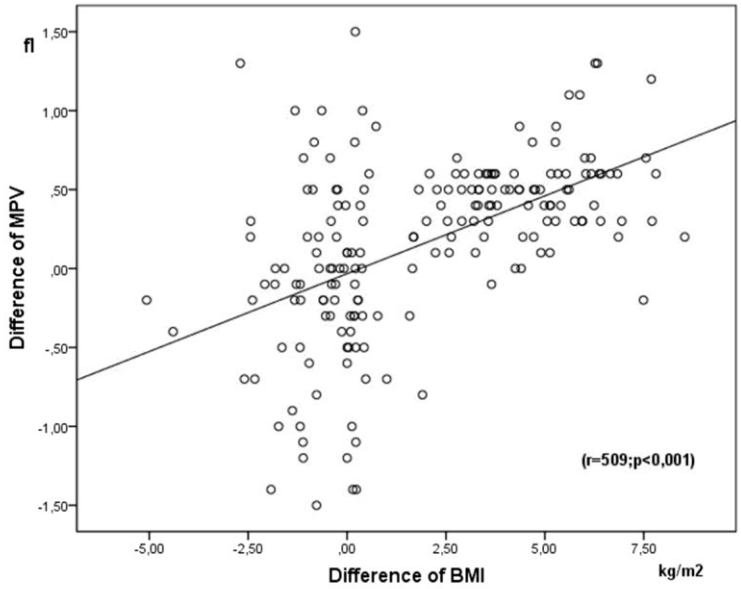

Figure 1: The correlation between the initial and final body mass index and mean platelet volume values

mean BMI2 of CG (35.20 ± 5.19) were compared, no significant difference was found (p> 0.05).

While no difference existed in the initial and final lipid parameters of CG (p>0.05), a significant difference existed in the initial and final lipid parameters of WLG (p<0.0001) (Table 1). A positive correlation existed between MPV and BMI values of all individuals in the study (r = 0.140; p =0.05). When this correlation was examined separately for both groups, it was found that there was a significant difference between BMI1 and MPV1 in WLG (r =0.267; p <0.01), whereas no difference was found in CG.

While no difference existed in the initial mean MPV values of each group, a significant difference was found at the end of the study (p <0.001). The reason of this result was to be decrease of MPV in WLG at the end.

It was discovered through univariate covariance analysis test that the change in initial and final measurement of MPV values of each individual in WLG was different from the change in CG (p <0.01).

There was a statistically significant correlation between the initial and final MPV and BMI values of individuals in the study (r = 0.509; p <0.001) (Figure 1). No correlation was found between NLR and BMI for both WLG and CG. Additionally, there was no correlation between weight loss and NLR (p> 0.05).

Discussion

In previous studies, it was stated that the level of obesity and MS are associated with inflammation and thus cause atherosclerosis (16). MPV which indicates platelet activity is associated with the pathological conditions in thrombopoiesis (16, 17). MPV levels in obese individuals are higher than non-obese individuals.

Coban et al. demonstrated in their study that MPV is higher in obese individuals than non-obese individuals (11). The results of studies examining the relationship of MPV with obesity and MS are incompatible with each other.

In a comprehensive study conducted by Kutlucan et al., it was found that MPV values of obese individuals with MS were different from values of obese individuals without MS (18). They claimed that MPV levels are more affected by obesity than MS. On the other hand, Tavil et al. stated that MPV is higher in MS and the reason for this was that it is related to HT, waist circumference, BMI, and fasting plasma glucose (19).

In literature, there are many studies in parallel with our study investigating the impact of diet on MPV in obese individuals. In the study conducted by Toplak et al., weight loss with hypocaloric diet and low calorie diet was examined. It was found in that study that MPV values increased in the first 8 weeks compared to the initial value, and MPV levels of individuals losing weight after 48 weeks dropped to the initial level. MPV did not change compared to initial level and no change was observed in lipid values (20). In our study, it was found that MPV and lipid values decreased after weight loss. When we compare our study and the study conducted by Toplak et al., it can be said that lipid levels have an important role in the effect of weight loss on MPV levels. In a study conducted by Coban et al., obese and non-obese individuals were compared. They found that MPV values in obese individuals were higher than non-obese individuals, and MPV values measured after weight loss whit diet decreased compared to the initial MPV values (21). However, the non-existence of control group consisted of obese individual without weight loss is the major limitation of that study. Unlike those studies, we investigated the changes in MPV levels of two groups including individuals with weight loss and without weight loss following the same diet. It was discovered that MPV levels decreased in obese individuals even if obesity continued after at least %5 weight losses.

Positive correlation existed between BMI and MPV values of individuals who participated in the study at the beginning. When this correlation is examined separately for both groups, it was discovered there was a significant correlation between BMI and MPV in WLG. However, no correlation existed between BMI and MPV in CG. Coban et al. stated that there was a significant correlation between BMI and MPV levels of obese individuals who lost weight, which is similar to our results (21). In addition, we found a positive correlation between the initial and final weight changes of individuals and the initial and final BMI changes of individuals (Figure 1). Accordingly, it can be said that MPV values decrease when individuals lose weight. While the mean MPV value of both groups was similar, the mean BMI value of WLG was higher than CG. At the end of the study, the mean BMI value of both groups was similar due to weight loss in WLG. Yet, the mean MPV value of WLG was lower than the mean MPV value of CG. Even if the mean BMI was similar, the likely reason of declining in MPV could be the decrease in lipid level of WLG. These findings suggest that MPV, which is an atherosclerotic marker in obese individuals, are more affected from lipid changes than weight loss.

There is no consensus about the use of NLR as a marker similar to MPV. NLR is an indicator of systemic inflammation, and there exists several studies asserting that it provides survey estimation after coronary intervention (22) and coronary artery bypass grafting operations (14). In the study conducted by Ryder et al., they found that NLR was not a useful marker in obese individuals (23). When Agacayak et al examined NLR in obese individuals with polycystic ovary syndrome (PCO), and obese individuals without PCO, they discovered that NLR was higher in obese individuals with PCO and they stated that high NLR might be a contributor in the formation of cardiovascular disease in individuals with PCO (24).

Bahadır et al claimed that NLR was lower in individuals who had a low BMI level than individuals who had a high BMI level. But it was not statistically significant (25). Additionally, in that study, it was reported that NLR , itself, could not be a reliable marker of inflammation, and did not differ according to the degree of obesity (25). Similar to that study, we found that there was no difference between the initial NLR value and the value after weight loss and NLR was not affected by BMI.

The present study had several limitations. First, design of the study is retrospective study. Obese individuals with no chronic diseases can investigate with prospective studies in the future. Second, our findings are based only on the Turkish population; different results might be observed in other ethnic groups.

Conclusion

As a conclusion even if obesity continues after at least 5% weight loss, MPV levels reduce. The changes in MPV values are profoundly affected by the changes of lipid values.

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

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