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Introduction:

Obesity is a chronic metabolic disorder associated with cardiovascular diseases and increased morbidity and mortality. Platelets are circulating, disc-shaped cells and their main role is to maintain the integrity of blood vessels through adequate hemostasis [2]. Circulating platelets may differ in size and hemostatic potential [2,3]. Larger platelets produce greater amounts of vasoactive and prothrombotic factors. They aggregate more rapidly under the stimulus of agonists and finally, they express a greater number of adhesion molecules leading to greater hemostatic efficiency [4,5].

Mean platelet volume (MPV), the most commonly used measure of platelet size, is a potential marker of platelet reactivity. Although there is still uncertainty about the most precise methodology for measuring MPV, it is routinely available in the inpatient and outpatient settings at a relatively low cost. Larger platelets are metabolically and enzymatically more active [6], and have greater prothrombotic potential [7]. Several reports have demonstrated that there is a close relationship between MPV and obesity, metabolic syndrome, body fat, and also weight reduction [8-9].

Aim:

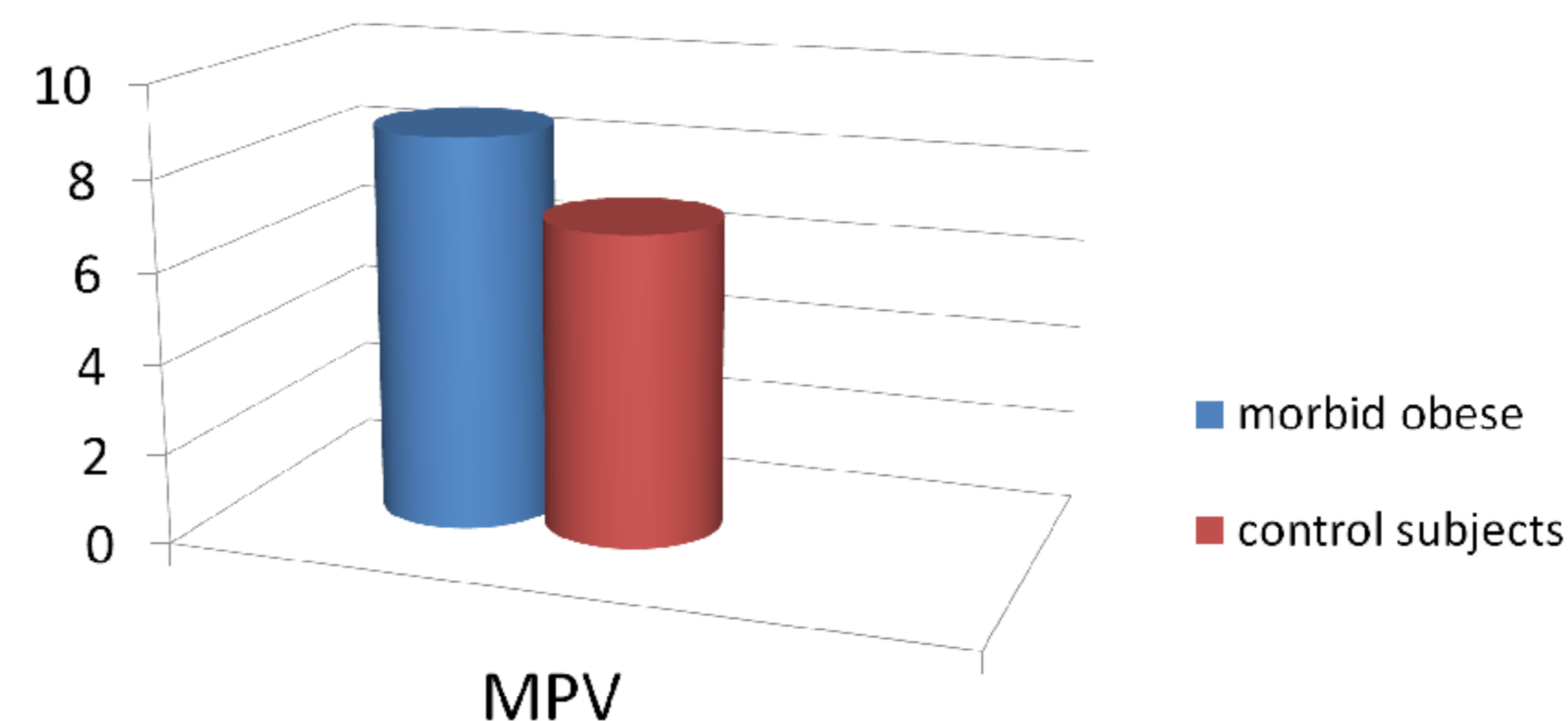
The incidence of atherosclerotic vascular disease is high in obesity. Our aim is to determine the mean platelet volume (MPV) as a new indicator of atherosclerosis in morbid obesity.

Material and Method:

Thirty-three patients with morbid obesity who applied to our endocrinology department (mean age 41.5 11.3 years, body weight 118.2 16.9 kg and body mass index (BMI): 45.9 7.2 kg/m²) and 39 age-matched (mean age 35.3 13.1 years; body weight 60.4 11.3 kg; BMI: 21.6 2.6 kg/m²) healthy individuals were included in the study.

Patients have hematological and other endocrinologic diseases including diabetes mellitus were excluded from the study. All complete blood count analysis was performed by automatic analyzer.

Results:



We found that mean MPV values and platelet counts in morbid obesity group were higher than control subjects (8.7 1.1 and 6.9 0.5 fL, $p=0.0001$; 267921.2 81475.3; 163710.8 13993.3 $\times 10^3/\mu\text{L}$, $p<0.0001$, respectively).

In addition, neutrophil lymphocyte ratio in morbid obesity group was not significantly different than those in control subjects (1.9 0.7 and 1.7 1.0, $p=0.225$, respectively). However, platelet to lymphocyte ratio was statistically different between groups (10730.5 58905.5 and 61614,7 58905.5 $p=0,001$ respectively). No statistically significant differences were found for the other parameters such as lymphocyte, WBC count and PCT.

There were positive correlations both between MPV and BMI ($r=0.649$, $p=0.0001$) and between MPV and body weight ($r=0.599$, $p=0.0001$).

Discussion:

In previously reports have been demonstrated that there was a close relationship between MPV and cardiovascular risk factors, such as impaired fasting glucose, diabetes mellitus, hypertension, hypercholesterolemia, obesity, metabolic syndrome, body fat, and also weight reduction [8-9]. Coban et al.[8] found that MPV was increased in obese patients and also showed a positive correlation with BMI level in the obese group without other cardiovascular risk factors. Our findings also suggest a similar interaction between morbid obesity and MPV.

Conclusions:

High MPV was associated with the presence of more metabolically active platelets. Therefore, increases of MPV and PLR in morbid obesity may lead to high risk for atherosclerosis.

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