The mean platelet volume in patients with obesity

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SUMMARY

Mean platelet volume (MPV), a determinant of platelet function, is a newly emerging risk factor for atherothrombosis. The present study was designed to evaluate MPV in patients with obesity compared with non-obese control subjects. We selected 100 non-obese subjects and 100 subjects with obesity [body mass index (BMI) ≥30 kg/m²] matched for age and gender. The MPV was significantly higher in obese group than in non-obese control group (10.3 ± 1.2 vs. 9.0 ± 0.8 fl, p < 0.01). MPV was positively correlated with BMI in obese group (p < 0.05). Increased MPV may be a possible cause for increased cardiovascular risk in patients with obesity.

Keywords: Cardiovascular risk; obesity; mean platelet volume

INTRODUCTION

Coronary heart disease is the major cause of death in the developed world (1). Platelet activation and aggregation are central processes in the pathophysiology of coronary heart disease (2,3). Mean platelet volume (MPV) is an indicator of platelet activation (4). It is increased in acute myocardial infarction, acute ischaemic stroke, preeclampsia and renal artery stenosis. Importantly, an elevated MPV predicts a poor outcome following myocardial infarction, restenosis following coronary angioplasty and the development of preeclampsia (5).

Obesity is a chronic metabolic disorder associated with cardiovascular disease and increased morbidity and mortality (6). MPV is increased in certain vascular risk factor states, including hypercholesterolemia, diabetes mellitus and hypertension (5,7–10). However, specifically, there has been no study about MPV in patients with obesity. Therefore, the present study was designed to evaluate MPV in patients with obesity compared with non-obese control subjects.

PATIENTS AND METHODS

Patients

We selected 100 non-obese normal subjects and 100 patients with obesity [body mass index (BMI) ≥30 kg/m²] matched for age and gender. Exclusion criteria for entry into the study were smoking habit, sustained hypertension (systolic blood pressure ≥140 mmHg and/or diastolic blood pressure ≥90 mmHg in clinic and daytime ambulatory), diabetes mellitus (fasting glucose 126 mg/dl or haemoglobin A1c >6.2%), hypercholesterolaemia (serum LDL cholesterol ≥160 mg/dl), hypertriglyceridaemia (serum triglyceride ≥400 mg/dl), renal failure (serum creatinine >1.5 mg/dl and blood urea nitrogen >30 mg/dl), heart failure, peripheral vascular disease, haematological disorders, acute or chronic infection, cancer and hepatic disease. Smokers and non-smokers were grouped according to their current smoking status. The BMI was calculated as the weight (kg)/height squared (m²). All patients gave their informed consent to participate in the study.

Biochemical Measurements

Blood samples were drawn after a fasting period of 12 h. Glucose, creatinine, alanine aminotransferase and lipid profile were determined by standard methods. We measured MPV in a blood sample collected in citrate (1:4 v/v) in order to avoid the platelet swelling induced by EDTA. A Cell-Dyn 3500 (Abbott) was used for whole blood counts.

Statistical Analysis

Statistical analysis was done by SPSS statistical software (SPSS for windows 10.0, Inc., Chicago, IL, USA). Data are expressed as mean ± SD. Groups were compared with Student’s t-test. Pearson’s correlation was used to evaluate the association between MPV and BMI. Statistical significance was defined as p < 0.05.

RESULTS

The main characteristics of study population are reported in Table 1. Age and gender distribution did not differ among
Table 1 Clinical and laboratory parameters in study groups

| Parameters                              | Obese group | Non-obese group | p    |
|-----------------------------------------|-------------|-----------------|------|
| Sex (males/females)                     | 38/62       | 37/63           | ns   |
| Age (years)                             | 49.3 ± 11.6 | 48.9 ± 11.8     | ns   |
| BMI (kg/m²)*                            | 32.7 ± 4.1  | 23.4 ± 4.3      | <0.01|
| Fasting glucose (mg/dl)                 | 90.6 ± 9.2  | 89.3 ± 9.4      | ns   |
| Creatinine (mg/dl)                      | 0.8 ± 0.2   | 0.8 ± 0.2       | ns   |
| ALT (U/l)                               | 28.8 ± 4.7  | 28.6 ± 4.6      | ns   |
| Total cholesterol (mg/dl)               | 187.1 ± 31.3| 186.4 ± 30.9    | ns   |
| LDL cholesterol (mg/dl)                | 114.1 ± 22.5| 110.9 ± 21.4    | ns   |
| HDL cholesterol (mg/dl)                | 48.4 ± 12.7 | 49.1 ± 12.9     | ns   |
| Triglyceride (mg/dl)                    | 133.7 ± 31.8| 132.9 ± 32.3    | ns   |
| Platelet counts (×10³/l)               | 287.4 ± 73.6| 286.3 ± 74.1    | p > 0.05 |
| MPV (fl)*                               | 10.3 ± 1.2  | 9.0 ± 0.8       | <0.01|

ALT, alanine aminotransferase; BMI, body mass index; HDL, high-density lipoprotein; LDL, low-density lipoprotein; MPV, mean platelet volume; ns, not significance; *p < 0.05, correlation between body mass index and MPV in obese group.

the groups by selection. Metabolic parameters were not different among the study groups as a result of the selection process. The BMI was higher in obese group than in control group (p < 0.01).

The MPV was significantly higher in obese group than in control group (10.3 ± 1.2 vs. 9.0 ± 0.8 fl, p < 0.01). MPV was positively correlated with BMI in obese group (p < 0.05). No significant difference was observed between obese and non-obese groups regarding platelet counts (287.4 ± 73.6 × 10³/l vs. 286.3 ± 74.1 × 10³/l, p > 0.05) (Table 1).

DISCUSSION

This is the first study, to our knowledge, to evaluate MPV in obese patients. In this selected population, we have found higher MPV in obese group than in control group, and no significant difference was observed between obese and non-obese groups regarding platelet counts. Little is known about the relation between BMI and MPV levels in obese patients. Toplak and Wascher (11) reported that after weight loss, the MPV was significantly decreased to initial values. In our study, we found that MPV showed positive correlations with BMI level in obese group.

The mechanisms for increased MPV as an indicator of platelet activation, in patients with atherosclerosis has not been elucidated. It may be speculated that three factors may contribute to increased platelet activation: (i) arterial wall injury; (ii) circulating inducers of platelet activation; and (iii) genetic predisposition (12–14).

We excluded patients with clinically overt cardiovascular disease (such as coronary artery disease, cerebrovascular disease and renal failure) to clarify the specific levels of BMI-related abnormalities. For this reason, our results cannot be extrapolated to all subjects with obesity. However, regardless of the mechanism, higher MPV represents a risk factor for atherothrombotic disease. Thus, this limitation does not lessen the clinical relevance of our results.

In conclusion, our data showed that middle-aged obese subjects without other cardiovascular risk factors have higher MPV levels than non-obese subjects. Increased MPV may be a possible cause for increased cardiovascular risk in patients with obesity.

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