

THE IMPORTANCE OF MEAN PLATELET VOLUME IN CORONARY ATHEROSCLEROTIC HEART DISEASE*

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SUMMARY : 40 patients with acute myocardial infarction (AMI) or unstable angina pectoris were included in this prospective study. All of the 40 patients were hospitalized at the 1st, 2nd, 3rd internal medicine clinics and coronary intensive care unit of Cardiology clinic of İzmir Atatürk State Hospital, between 1st of January and 31st of March 1992. Other 20 patients who came to the outpatient clinic with non-cardiac complaints were chosen as control group. There wasn't any recognized major risk factors in any of the patients in the control group and resting ECG's were normal. Mean Platelet volumes (MPV) were measured for all patients. Median MPV was 9.46 ± 0.14 fl in AMI group; 9.42 ± 0.2 fl in unstable angina pectoris (UAP) group and 8.15 ± 0.14 fl in control group. MPV differences between patients and controls were statistically significant.

As a result we concluded that MPV should be accepted as a risk factor in coronary atherosclerotic heart disease.

Key Words : Mean Platelet Volume, Coronary Artery Disease.

INTRODUCTION

Platelets have an important role in atherosclerosis and development of thrombosis (13, 15, 16). The aim of this study was to determine the effect of platelet volume on the development of coronary atherosclerotic heart disease. In order to do this; we planned to compare the MPV of the acute myocardial infarction and unstable angina pectoris patients with the control group consisting of non cardiac patients.

MATERIALS AND METHODS

40 patients with acute myocardial infarction (AMI) or unstable angina pectoris (UAP) were included in this prospective study. All of the 40 patients were hospitalized at the 1st, 2nd, 3rd internal

medicine clinics and coronary intensive care unit of Cardiology clinic of İzmir Atatürk State Hospital, between 1st of January and 31st of March 1992. Diagnosis of the AMI and UAP of the hospitalized patients in the first two groups was supported by clinical, enzymatic and electrocardiographic findings. Ages of the AMI and UAP patients were between 38 and 71, with an average of 56. Thirty two of these patients were male (80 %).

Control group was consisting of 20 patients who came to the outpatient clinic for non cardiac complaints. There wasn't any risk factors such as cigarette smoking, high blood pressure, diabetes mellitus and hyperlipidemia in these 20 patients. None of these 20 patients suffered from angina pectoris and resting ECGs were normal. Ages of the control gro-

up were ranging between 32 and 58 with an average of 45. Sixteen of the control group were male (80 %) (Figure 1).

In all of the AMI and UAP patients blood samples were taken for enzymatic analysis after the diagnosis was established by the means of clinical and electrocardiographical examination. 12 hours after the onset of chest pain blood samples were taken to measure MPV.

In all of the patients of the control group blood samples were taken to analyse cholesterol, triglyceride and MPV values, after clinical and electrocardiographical examination.

According to the calibration of the instrument (MEDONIC CELL ANALYZER CA 600) normal range of MPV was between 7 and 11 femtoliters with an average of 9 femtoliters.

RESULTS

There wasn't any differences in male/female rate between the study and control groups. 26 of the 40 patients in the study group were AMI (65 %), and 14 were UAP (35 %) (Figure 1). 21 of the 26 AMI patients (52.5 % of total) and 11 of the 14 UAP patients (27.5 % of total) were male while 8 of the 40 patients were female. 5 of these female patients were AMI (12.5 % of total), and the remaining three were UAP (7.5 % of total) (Figure 2).

Ages of the 60 percent of the study group were between 45-59, 30 percent were over 60 and 10 percent were between 30-44. On the other hand 80 percent

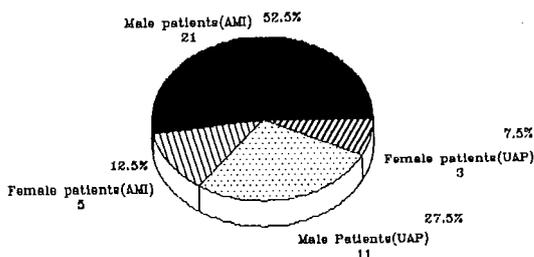


Fig - 2 : Patient group with coronary atherosclerotic heart disease.

cent of the control group were between 30-44 and 20 percent were between 45-59. Median age was 56 ± 1.47 (38-71) in the study group and 45 ± 1.88 (32-58) in the control group. There was a significant difference between the age distribution of study and control groups ($p < 0.01$).

MPV values were 8 fl in 15.3 %, 9 fl in 42.3 %, 10 fl in 26.9 %, 11 fl in 11.5 % and 12 fl in 4 % of the AMI patients of the study group.

Among the UAP group, MPV values were 8 fl in 7.1 %, 9 fl in 50 %, 10 fl in 35.8 % and 11 fl in 7.1 % of these 14 patients. In control group MPV values were 7 fl in 15 %, 8 fl in 55 %, 9 fl in 30 % of the 20 patients (Figure 3).

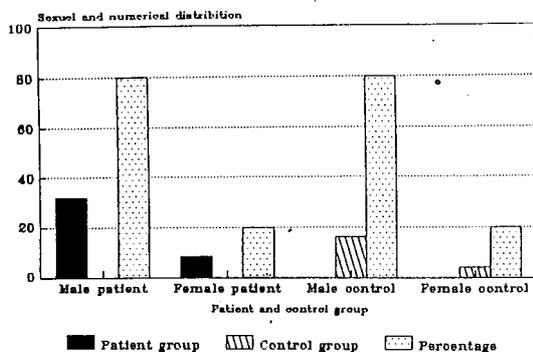


Fig - 1 : Sexual and numerical distribution of patient and control groups.

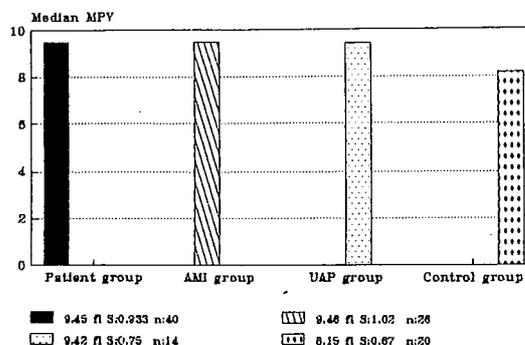


Fig - 3 : Median MPV values in patient and control groups.

Median MPV values of the study and control groups are shown in Figure 4. Median MPV values were 9.45 ± 0.14 fl and 8.15 ± 0.14 fl in study and control groups respectively. The difference between these values was statistically significant ($p < 0.01$). The differences between the median MPV values of the AMI patients and the control group, and median MPV values of the UAP patients and the control group were also found to have a statistical significance separately ($p < 0.01$). But the difference of the median MPV values of the AMI patients and UAP patients wasn't significant statistically ($p > 0.01$).

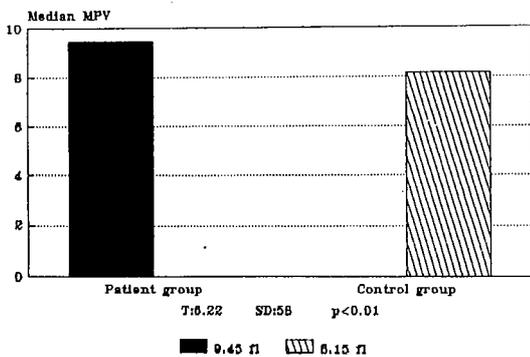


Fig - 4 : Median MPV values of patient group in comparison with control group.

DISCUSSION

Platelets may have an important role in developing of atherosclerosis as the conclusion of various experimental studies on laboratory animals and a research on Greenland Eskimos (3,4). It is almost certain that platelets have a role in occurrence of atheromatous coronary artery, however that process is multifactorial. Platelets probably have the role on mitogen secretion from alpha granules or other products (7).

Platelets contain some substances such as serotonin, epinephrine, thromboxane A2, C-endoperoxides, thromboxane B2, cationic proteins, mitogenic factors (immigration and proliferation of endothelium, vascular smooth muscle and fibrosis), beta tromboglobulin, lysosomal enzymes and von Willebrand factor and these substances have possible importance on thrombosis and atherosclerosis (1).

The mean age of our control group was lower than the study group. But Von Bahren showed that human platelet volume doesn't increase with aging (17). So we believe that this difference in our study doesn't make our results or conclusions unreliable.

MPV values of the AMI patients in our study were found higher than controls as in the research of Martin et al (10). MPV values in the UAP patients were also higher than controls but we couldn't find enough research on the MPV values in UAP patients (16). Kishk et al. found that MPV values in AMI patients were significantly higher than the patients who have heart disease but no infarct and the asymptomatic group (8).

It has been thought that there are three reasons of increase of platelet volume before AMI (10).

The first, volume increase which has been detected during the first 12 hours of the hospitalization reminds us that this increase had already been present before the onset of the disease. Because life time of platelets is approximately eight days and more than 90 % of platelets which was counted immediately after AMI had already been in circulation before the vessel occlusion.

The second reason to think, the mean platelet volume in AMI group had been higher for a long time as the continuing high values 6 weeks after discharge from hospital despite the mostly healed infarcts.

The third and the last; by adding the continuity of logarithmic normality of the platelet volume in study group to discussion, it was observed that increased volume hadn't been occurred as a result of occurrence or consumption of a variety of platelet subgroups, and that all distribution curve has been changed.

Trip et al. measured spontaneous platelet aggregation in 149 AMI patients and followed these patients up to five years (18). Positive results (spontaneous aggregation in 20 minutes) had a stronger correlation with deaths than negative results. Therefore it seemed possible to determine the risk of nonfatal reinfarct or death by showing the presence or absence of positive spontaneous platelet aggregation, the increased platelet releasing reaction and the existence of big and more reactive platelets in post myocardial infarction cases. The reactivity of the platelet increases with the volume of itself (11, 14).

Martin and co-workers measured MPV as a criteria of platelet reactivity in 1716 patients 6 months after AMI (14). Throughout the 2 years follow up they evaluated recurrent ischemic heart diseases and deaths. MPV values were higher in the 126 patients who had a second ischemic attack (fatal or non-fatal) during the follow-up period than the 1590 patients who didn't experience a second attack ($p<0.01$). On the other hand, among the 126 patients who had a second attack, MPV values of the dead patients were higher than the survivors ($p<0.01$). There wasn't any differences of platelet counts between these groups. When MPV was evaluated with the age of the patients, it was noted that there was a tendency of fixed increase for death and re-infarct. On the other hand, there wasn't any correlation between MPV and known ischemic heart disease such as blood pressure, blood lipids, leucocyte count and plasma viscosity.

The increase of MPV and shortening of bleeding time were clear in AMI patients when compared to control group. Also the size and DNA content of the megakaryocytes have been found to be increased (2, 6, 9, 12).

Kishk and co-workers measured MPV and platelet counts in three groups. The first group was consisting of AMI patients, the second group was patients with myocardial ischemia but no infarct and the third group was young asymptomatic males (8). MPV values were higher in the AMI group compared with ischemic and asymptomatic groups and the differences were statistically significant.

It is well understood that big platelets are an independent risk factor for AMI, because there is no relation between MPV and other risk factors. Probably the control of platelet production from megakaryocytes carries a therapeutic potential for AMI at the future (5, 9).

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