

The state of platelets and their functional activity in patients with arterial hypertension in combination with non-alcoholic fatty liver disease

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Background: According to modern concepts of hypertension (HT) and obesity, the hemostatic system in these diseases is characterized by pro-thrombogenic changes. Since the liver is the site of the formation of many factors of hemostasis, the concomitant non-alcoholic fatty liver disease (NAFLD), which often accompanies the course of both of these diseases, is an actual problem.

Purpose: To improve the efficiency of early diagnosis of thrombophilic blood changes in hypertensive patients with concomitant obesity and NAFLD by determining the state of platelets and their functional activity.

Materials and methods: We examined 167 patients (80 men and 87 women). The average patient age 55.5 [47.0; 61.0] years. Patients were divided into three groups: I - 46 patients with hypertension without NAFLD, II - 54 patients with NAFLD without hypertension, III group - 52 hypertensive patients with NAFLD. The control group consisted of 15 healthy subjects matched for age and sex. Platelet count and mean platelet volume (MPV) was performed on an automated analyzer, spontaneous and induced platelet aggregation was evaluated by laser aggregometer.

Results: A significant increase in the degree of spontaneous aggregation of platelet has been found in patients in all groups compared to the control group: I group - 2.2-fold increasing in aggregation ($p < 0.05$), II group to 4.2-fold ($p < 0.05$), III group had increasing by 4.1-fold ($p < 0.05$).

ADP-induced platelet aggregation was the same in I group and control, but it was 39% higher ($p < 0.001$) in II cohort and by 22.6% ($p < 0.01$) in III group.

Adrenalin-induced platelet activity increased in all groups versus control: I group – 2.1-fold ($p < 0.001$), II group – 2.3-fold ($p < 0.001$), III group – 1.6-fold ($p < 0.01$) elevation.

Arachidonic acid-induced aggregation elevated by 64.2% ($p < 0.001$) in I group and decreased by 56.3% ($p < 0.01$) in II and by 43% ($p < 0.05$) III cohorts.

Collagen-induced activity had not significant difference between groups. MPV was increased in both groups with NAFLD by 5.9, but in II group significance level was higher - $p < 0.01$ than in group III - $p < 0.001$. MPV had not significant changes in I group versus control.

Conclusion: Spontaneous platelet aggregation is increased in hypertensive patients and it is significantly enhanced in combination with NAFLD. Thus in patients with isolated NAFLD also observed a statistically significant increase in spontaneous aggregation of platelets. That is possibly explained by an increase in MPV which can be considered as one of the NAFLD risk factors thrombophilic changes in primary hemostasis. An analysis of induced platelet aggregation showed that patients with NAFLD may have a lower sensitivity to acetylsalicylic acid therapy.