State of platelet-plasma hemostasis in patients with hypertension and non-alcoholic fatty liver disease in condition of hypercholesterolemia

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Non-alcoholic fatty liver disease (NAFLD) is a marker of metabolic and dyslipidemic disorders. Given the prevalence of atherogenic dyslipidemias and their proven effect on the development of thrombotic cardiovascular complications in patients with NAFLD, it is important to understand the role of hemostatic blood activity.

Aim of the study: To increase the effectiveness of early diagnosis of thrombophilic blood changes in patients with hypertension (HT) combined with non-alcoholic fatty liver disease by determining the state of plasma hemostasis in conditions of hypercholesterolemia and concomitant statin therapy.

Materials and methods: 152 patients were examined. Groups of patients: I – 46 patients with stage II GC, II – 54 patients with NAFLD without GC, group III – 52 patients with stage II GC with concomitant NAFLD. The analysis of spontaneous and induced platelet aggregation, indicators of coagulation, anticoagulant and fibrinolytic hemostasis.

Results: The degree of spontaneous aggregation was significantly higher in patients with hypercholesterolemia – by 32.4% ($p<0.05$). The level of fibrinogen was higher by 13.5% ($p<0.05$) due to hypercholesterolemia, at the same time there was a decrease in antithrombin (AT) III by 8.7% ($p<0.05$) in patients with high cholesterol. Patients with comorbid HT and NAFLD on statin therapy had a 16.5% ($p<0.05$) lower degree of spontaneous aggregation than patients who did not receive this treatment. In the NAFLD group, patients receiving statins had a significantly lower degree of arachidonic acid-induced aggregation against patients without lipid-lowering therapy (by 54.0%, $p<0.001$). However, the analysis of the general population of the examined revealed a decrease in the degree of collagen-induced aggregation by 38.7% ($p<0.05$) in a subgroup of patients receiving treatment. We observed a 23.4% ($p<0.001$) shortening of prothrombin time in the NAFLD group and a 16.0% ($p<0.05$) shortening in the combined course of HT and NAFLD. In the absence of statin therapy in the NAFLD group, there was a significant decrease in INR – by 9.7% ($p<0.05$) – compared with patients receiving lipid-lowering therapy. A decrease in thrombin time by 12.2% ($p<0.05$) was observed in the subgroup receiving statins among patients with NAFLD. In the general cohort, the use of statins increased the activity of AT III by 10.7% ($p<0.01$), but in the NAFLD group this difference was more significant – by 14.3% ($p<0.001$) AT III was more active in patients who received lipid-lowering therapy.

Conclusion: The results of the analysis showed that patients with hypercholesterolemia have procoagulant and prothrombogenic activity of blood. But statin treatment decrease platelet aggregation, blood coagulation potential and increased activity of anticoagulant hemostasis, which complements the mechanism of prophylactic effect of HMG CoA reductase inhibitor (statin).