Influence of heart rate variability on platelet hemostasis by average aggregate size in patients with chronic coronary heart disease in combination with COVID-19

V.Z. Netiazhenko[1], S.Ye. Mostovyi[1, 2]
1. Bogomolets National Medical University, Kyiv, Ukraine
2. Medical Centre "Medbud", Kyiv, Ukraine
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BACKGROUND. In order to prevent complications of coronavirus infection (platelet hemostasis disorders, heart rate variability and QT interval dispersion, which increase the risk of thromboembolic complications and fatal arrhythmias), it is promising to study the relationship between heart rate variability and platelet hemostasis in patients with coronary artery disease (CAD) in combination with coronavirus disease (COVID-19).

OBJECTIVE. The aim of the study was to investigate the impact of COVID-19 on the interdependence of changes in platelet hemostasis and heart rate variability in patients with CAD.

MATERIALS AND METHODS. We examined 102 patients divided into three groups: group 1 – CAD without COVID-19 (n=32); group 2 – CAD in combination with COVID-19 (n=35); group 3 – COVID-19 without CAD (n=35). The control group included 30 conditionally healthy individuals. Changes in platelet hemostasis were studied according to laser aggregometry by the Born method and analysis of the average size of aggregates with an assessment of spontaneous aggregation and aggregation induced by adenosine diphosphate (ADP), arachidonic acid, epinephrine, collagen, ristomycin. Heart rate variability parameters and QT interval variability characteristics were determined by the results of 24-hour Holter electrocardiogram monitoring.

RESULTS AND DISCUSSION. The time for spontaneous aggregation was longer in all patients compared to the control group, with the highest values observed in groups 2 and 3 (with COVID-19). In ADP aggregation, the control group showed higher results. The rate of aggregation in ADP was lowest in group 2. Collagen-induced aggregation was higher in COVID-19 cases. All groups exhibited lower values of adrenaline-induced aggregation, with the lowest values in group 3. The time of aggregation under adrenaline was shortest in group 2. Ristocetin induced greater aggregation in the control group, but the lowest rate in group 2, and the shortest time in group 1 (CAD without COVID-19). In CAD with COVID-19, an inverse relationship was observed between the parasympathetic nervous system and adrenaline-induced aggregation, as well as between sympathetic activity and ristocetin-induced aggregation.

The degree of platelet aggregation induced by ADP had a direct correlation with the corrected QT interval. The rate of aggregation with ADP had an inverse correlation with the mean QT. The time of platelet aggregation activated by ristocetin had an inverse correlation with the standard deviation of QT(NN). The mode of QT had an inverse correlation with the degree of aggregation with ristocetin.

CONCLUSIONS. In CAD and COVID-19, platelet hemostatic function is impaired, as confirmed by laser aggregometry data. Autonomous dysregulation and prolonged QT interval have been identified. A complex interaction between platelet hemostasis and heart rate variability is observed. Considering platelet hemostatic function, heart rate variability, and QT dispersion is crucial for treating this cohort of patients.

KEY WORDS: chronic coronary heart disease, COVID-19, heart rate variability, QT interval variability, platelet aggregation, laser aggregometry parameters by average aggregate size.