Evaluation of the relationship between anisocytosis and the quantitative and qualitative characteristics of coronary atherosclerosis.

Despite the increasingly sophisticated methods of diagnosis and treatment, coronary artery disease remains the most common single cause of death in developed countries. In Europe - 1.8 million deaths each year. It is estimated that every year 0.3% of the population is experiencing an acute coronary syndrome, with an annual mortality and post-infarction heart failure at 12% and 24% respectively.

The aim of the study is to clarify whether there is a link between anisocytosis (expressed as *RDW, Red Cell Distribution Width*) and quantitative (*plaque burden*) and qualitative (e.g. the amount of lipid component, vascular remodeling) parameters of atherosclerotic plaques in the coronary arteries. RDW is an indicator of heterogeneity in the size of circulating erythrocytes. In a recently published original study we showed that the RDW is an independent strong predictor of death and myocardial infarction in patients with coronary artery disease. Despite the increasing number of data confirming the prognostic value of anisocytosis in patients with coronary atherosclerosis (including patients with stable coronary artery disease, acute coronary syndrome, heart failure due to ischemia), the pathophysiology of this association remains unknown. Some authors suggest that the presence of anisocytosis may reflect chronic subclinical inflammation, oxidative stress and impaired erythropoiesis. Recent reports, however, does not support these suggestions. We hypothesized that a potential patomechanism linking anisocytosis with the occurrence of cardiovascular events may be: RDW relationship with extent of coronary atherosclerosis and/or with the presence of high risk atherosclerotic plaques in the coronary arteries. This hypothesis has not yet been investigated.

The project will examine the potential pathophysiological mechanisms of anisocytosis connection with worse prognosis in patients with coronary artery disease, according to the best of our knowledge, for the first time taking into account potential confounders such as active inflammation, microcytic/megaloblastic anemia. Clarification of these pathophysiological issues can be a starting point for prospective clinical trials aimed at developing more effective diagnostic algorithms in patients with known coronary artery disease. This may be of great importance for risk stratification in this group of patients, allowing for individualization of therapeutic intervention.