Atherosclerotic plaque leads to coronary artery disease and its life-threatening complicances, i.e. myocardial infarction. The process of the plaque development is very complex and depends on different intracellular pathways. There are pathways which promote atherosclerotic plaque progression (i.e. atherogenesis), but on the other hand there are factors that causes inhibition of this process and protection from atherosclerosis, for instance proteins KLF2, KLF4, MertK, IL-10 and TGF-β3. These proteins acts upon different points of atherogenesis like extracellular structure change, vascular inflammation and cellular death. The aforementioned factors are negatively regulated by microRNA particles, which inhibit genes expression by binding to mRNA of the proteins and causing their degradation. Owing to microRNA bioinformatic database containing microRNA particles and their target proteins – miRTarBase 7.0, I have selected specific microRNA particles, i.e. hsa-miR-92a-3p that inhibits KLF2, hsa-miR-10b-5p that inhibits KLF4; hsa-miR-126-3p that inhibits MertK, hsa-miR-98-5p that inhibits IL-10 and hsa-miR-29b-3p that inhibits TGF-β3. We assume, that these microRNA particles act as molecular switches and we hypothesize, that their concentration is increased in patients with more advanced coronary atherosclerotic plaque. In order to verify our hypothesis, I have designed the study, in which 60 patients admitted to Invasive Coronary Unit for coronary angiography for diagnosis and evaluation of coronary artery disease will be included. Blood samples will be taken from each patient and serum level of microRNA particles - hsa-miR-92a-3p, hsa-miR-10b-5p; hsa-miR-126-3p, hsa-miR-98-5p and hsa-miR-29b-3p will be measured. Next, the levels of these microRNA particles will be correlated with atherosclerotic plaque burden assessed by Gensini score. I hypothesize, that patients with more advanced coronary atherosclerosis (more Gensini score points) will present increased expression level of selected microRNA particles. The results of our investigation will enable us to discuss the role of certain atheroprotective intracellular pathways in coronary atherosclerotic plaque development and progression. If our study demonstrates significant and strong correlation between selected microRNAs expression and atherosclerotic burden, we will be able to formulate new hypothesis about steps in atherosclerotic plaque progression and design new potential methods of atherosclerosis prevention in the future.