Abstract

Atherosclerosis, or sclerosis of arteries, is a degenerative disease of arteries. Sometimes it is called „the disease of 20th century".

ApoE/LDL – receptor double knockout mice represent a new animal model for study of atherogenesis, which is characterized by severe hyperlipidaemia and atherosclerosis.

Statins (or competitive inhibitors 3-hydroxyl-3-methyl-glutaryl-coenzym A-reductase) currently belong to the most efficient and the most useful hypolipidemic drugs for all over the world. They decrease mainly levels of total cholesterol and LDL cholesterol.

The aim of this thesis was to describe the expression of endoglin in atherosclerotic plaques in apoE/LDL-receptor deficient mice. Moreover we wanted to determine endoglin colocalization with eNOS and the effect of atorvastatin treatment on the expression of both molecules.

ApoE/LDLR-deficient mice on were subdivided into 2 groups. The control group of animals was fed with the western type diet. The same atherogenic diet was used in ATV group, where atorvastatin was added to the atherogenic diet at the dosage of 100 mg/kg per day.

The results of this thesis confirmed the expression of endoglin in atherosclerotic lesions in ApoE/LDLR-deficient mice. The expression of endoglin was located on the aortic vascular endothelium and in other smaller vessels and capillaries of surrounding myocardium. Immunohistochemical analysis showed endoglin co-expression with eNOS in aortic endothelium in both control and atorvastatin treated mice. Atorvastatin treatment resulted in a strong hypolipidemic effect. Stereological analysis demonstrated that atorvastatin significantly increased expression of endoglin and eNOS in mice aorta.

In conclusion, we propose, that endoglin might play protective role in atherogenesis by increasing eNOS expression in aortic endothelium in apoE/LDLr-deficient mice.