Abstract

Non-alcoholic fatty liver disease (NAFLD) is a chronic disease of the liver tissue. More advanced stages of NAFLD are characterized by the development of fibrosis. The fibrotic process occurs at the stage of nonalcoholic steatohepatitis and is usually accompanied by inflammation.

The aim of this study was to analyze the expression of fibrosis markers (α - SMA, galectin - 3 and collagen). As part of the study design, mice were divided into two groups, namely a control group fed a standard laboratory diet (chow diet) and an experimental group fed a CDAA diet. CDAA diet leads to the development of fibrosis through inhibition of fatty acid oxidation in hepatocytes, increase in lipid synthesis and development of oxidative stress and inflammation. Mice were fed for 4 weeks. The expression of selected fibrosis markers was detected and evaluated by indirect immunohistochemistry using the ABC method.

The results of this thesis show that α-SMA marker positivity was stronger in the experimental model and was also detected in activated hepatic stellate cells and myofibroblasts in the liver. Galectin-3 staining was also more pronounced in the experimental group than in the control group. Sirius Red staining for the detection of collagen fibers was also more intense in the experimental group, where the analysis showed the presence of collagen outside the smooth muscle cells of blood vessels. All of these aspects suggest an ongoing fibrotic and inflammatory process after administration of the CDAA diet. In conclusion, fibrosis markers were increased in the experimental CDAA group.

Keywords: liver, immunohistochemistry, NAFLD, fibrosis, α-SMA, galectin 3, collagen