Streszczenie w języku angielskim

Glycosylation is one of the most common co- and posttranslational modifications of proteins. The *majority of blood proteins are glycosylated. The products of protein glycosylation are glycoproteins. They differ in their protein and carbohydrate structure, which is the basis for the occurrence of specific isoforms. T*here is abundant scientific evidence supporting changes in protein glycosylation in inflammatory conditions of various etiologies, resulting in quantitative alterations in glycoprotein isoforms. One of the highly variable glycoproteins is the negative acute phase protein - transferrin. It is characterized by microheterogeneity, which results from differences in the glycosylation of oligosaccharide chains. Depending on the number of attached sialic acid residues, there are 9 transferrin isoforms, the profile of which changes in inflammatory and non-inflammatory diseases.

The aim of the study was to determine the serum concentrations of transferrin isoforms in primary cholangitis (PBC) and acute (AP) and chronic (CP) pancreatitis. In the first study, we compared the concentration of total transferrin and its isoforms between a group of people with PBC, with extrahepatic cholestasis (to eliminate the effect of cholestasis on the change the profile of transferrin isoforms) and a group of healthy people. The concentrations of transferrin isoforms in patients with PBC were also assessed depending on the histological stage of the liver disease according to the Ludwig scale. In the second study, we compared the serum total concentration of transferrin and its isoforms between the groups of patients with pancreatitis and the control group. Additionally, the concentrations of transferrin isoforms depending on the morphological form and etiology of acute pancreatitis were assessed. Based on the literature, the profiles of transferrin isoforms in pancreatitis were compared with those in other diseases: pancreatic cancers, rheumatic diseases and chronic hepatitis.

The material for the studies was venous blood serum. The concentrations of transferrin isoforms were analyzed by capillary electrophoresis on the Sebia MINICAP system. The first study included 76 patients with PBC. Primary biliary cholangitis was diagnosed according to the guidelines of the European Association for the Study of the Liver (EASL). In addition, 44 people with extrahepatic cholestasis (Vater papilla cancer) and 40 healthy people were studied. The second study involved 84 patients with acute pancreatitis and 42 patients with chronic pancreatitis. The control group consisted of 30 healthy people

An increase in the total concentration of transferrin was observed in the serum of patients with PBC. Among the transferrin isoforms, di- and trisialotransferrin concentrations in patients with PBC were lower than in healthy people. There were no changes in the

concentrations of tetra- and pentasialotransferrin. The profile of transferrin isoforms in extrahepatic cholestasis was different: the concentrations of disialotransferrin and tetrasialotransferrin were higher than in PBC, whereas the concentration of pentasialotransferrin was not changed. There were no significant changes in the concentrations of transferrin isoforms depending on the histological stage of PBC as measured by the Ludwig scale.

In AP and CP, the concentration of total transferrin was significantly lower than in the group of healthy people. Moreover, in AP it was lower than in CP. The analysis of the profiles of transferrin isoforms showed a lower concentration of pentasialotransferrin in patients with AP and CP than in the control group. Additionally, in patients with acute pancreatitis, the concentration of tetrasialotransferrin was significantly higher than in healthy people. There were no significant differences in the concentrations of transferrin isoforms between the groups with AP and CP.

In conclusion, the determination of transferrin isoforms concentrations in liver and pancreatic diseases may provide additional information about the studied diseases. The profile of transferrin isoforms in PBC is characteristic of this disease but doesn't change with the severity of the disease. Changes in the profile of transferrin isoforms in acute and chronic pancreatitis are not specific for this organ, but relate to the pathogenesis of the disease. Furthermore, these changes are independent of the morphological form and etiology of acute pancreatitis. A comparative analysis of transferrin isoform profiles in various diseases may be important in the primary and differential diagnosis of these diseases