CLINICAL AND LABORATORY FEATURES OF THE COURSE OF UPPER GASTROINTESTINAL PATHOLOGY WITH CONNECTIVE TISSUE DYSPLASIA

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The **aim** of the investigation was to study the state of endothelial function, connective tissue metabolism in patients with upper gastrointestinal pathology against the background of connective tissue dysplasia to develop a management system of patients at the primary health care level.

Materials and Methods. The study enrolled 125 patients with upper GI tract diseases aged 20-55 years, mean age 32,5±13,8 years. Patients were divided into 2 groups: the first group included 45 (36%) patients with upper GIT diseases without signs of CTD, the second group included 80 (64%) patients with signs of CTD and 20 patients for the control group.

Results. Clinical studies by age and sex revealed that the disease was predominantly in the 30-40 age group (51.2%). We investigated some parameters of NO system in patients with upper gastrointestinal pathology against the background of CTD. Our investigations revealed 1,12 (P>0,05) and 1,34 (P<0,001) times increase of stable nitric oxide metabolites in patients of 1st and 2nd groups of investigation. It should be said that eNOS is responsible for nitric oxide synthesis by endothelial cells. The analysis of its level showed the tendency to a decrease in the group I patients, while in the group II patients these changes were statistically significant, decreasing 1.23 (P<0.001) times in comparison with the values of practically healthy persons. According to some researchers, low eNOS values in atherosclerosis and inflammation protect endothelial cells from apoptosis. At the same time, it can be assumed that some decrease in eNOS activity in serum is associated with an impairment of its production by endothelium due to endotheliocyte desquamation. Analysis of its activity showed a progressive increase with aggravation of the pathological process. While iNOS activity increased 1.2 (P>0.05) times in patients of Group I, also it was 1.36 (P<0.001) times higher in patients of Group II than in control group. The activation of iNOS, causing NO overexpression, is stimulated by activated oxygen species and proinflammatory cytokines. Analysis of serum peroxynitrite determination in patients revealed a 1.15 (P>0.05) and 1.31 (P<0.01) fold increase in its level in Group 1 and Group 2 respectively. The excess of NO and increased formation of highly cytotoxic $\hat{I}NO2^{-}$ by the feedback type depresses basal level of eNOS activity to a greater extent and initiates pathological NOS isoform - iNOS.

Thus, endothelial dysfunction due to an imbalance in the NO system is observed in patients of 1st and 2nd group. Free-radical processes in cells are under control of enzymatic and non-enzymatic AOS system. The leading role here belongs to the enzyme SOD, which prevents the accumulation of free oxygen radicals. Thus, SOD activity in 1st group patients increased statistically significantly up to 2,518±0.284 UU op/ml (P<0.01), while this parameter in healthy subjects was 1,997±0.161 UU op/ml. At the same time, the catalase activity in the blood serum of the 1st group patients was 29,27±0,90 μcat/l, while the value of this parameter in practically healthy persons was 21,90±0,67 μcat/l, which exceeded the normative values by 1,34 (P<0,05) times. The compensatory mechanisms of endothelium function regulation were evidently conserved in this group of patients. At the same time, in patients in Group 2 we observed a tendency to decrease the activity of SOD down to 1,800±0,085 UU/ml, the activity of catalase was still within the range of values in practically healthy subjects, equal to 20.59±0.68 μcat/l, testifying to the beginning of the compensatory mechanisms failure.

Thus, patients with GIT pathologies associated with CTD show decreased blood magnesium levels, hyaluronidase activation, which contributed to increased decomposition of extracellular matrix components with increased excretion of their decomposition products. Endothelial dysfunction due to an imbalance in the NO system was also noted. The imbalance in the NO system, in turn, is determined by iNOS hyperexpression and accumulation of ONO2-, inhibition of eNOS activity.

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