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TOXIGENIC STRAINS OF HELICOBACTERIAL INFECTION, HISTOLOGICAL CHANGES OF THE MUCOSA IN PATIENTS WITH DUODENAL PEPTIC ULCER AND TYPE 2 DIABETES MELLITUS

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The article presents an assessment of the prevalence of Helicobacter pylori infection strains and their influence on the duodenal mucosa in patients with peptic duodenal ulcer (PDU), including.

The study involved 48 patients with H. pylori-associated PDU, including 28 with concomitant T2DM. The control group consisted of 22 practically healthy individuals without acute or chronic diseases. Genotyping of H. pylori strains for the presence of cagA and vacA genes was performed, and histological and histochemical examinations of duodenal biopsy specimens were carried out.

In patients with isolated PDU, the distribution of H. pylori genotypes was as follows: $cagA^+vacA^+$ — 55%, $cagA^+vacA^-$ — 5%, $cagA^-vacA^+$ — 30%, $cagA^-vacA^-$ — 10%. In patients with PDU combined with T2DM, the $cagA^-vacA^+$ genotype occurred
4.67 times more frequently compared to $cagA^+vacA^+$ and $cagA^+vacA^-$ strains. Morphological evaluation revealed more pronounced endothelial dysfunction in patients harboring $cagA^+vacA^+$ strains — manifested by a higher proportion of vessels with endothelial desquamation (by 42.1% in isolated PDU, by 19.35% in PDU with T2DM; $p < 0.05$), reduced endotheliocyte nuclear volume (by 36% and 25.3%, respectively; $p < 0.05$), and increased variability of nuclear chromatin distribution (by 26.1% and 17.6%, respectively). The optical density of the PAS reaction was significantly reduced in $cagA^+vacA^+$ carriers, indicating impaired mucus formation, which was further aggravated in the presence of T2DM.

In patients with PDU and T2DM, the $cagA^-vacA^+$ genotype predominates, occurring 4.67 times more frequently than $cagA^+vacA^+$ and $cagA^+vacA^-$ strains. The presence of $cagA^+vacA^+$ genes is associated with more severe endothelial dysfunction — higher endothelial desquamation, reduced nuclear volume, and greater chromatin variability. These changes are more pronounced in patients with combined PDU and T2DM. Histological signs of inflammation (polymorphonuclear leukocyte infiltration, stromal edema, stasis, and hemorrhages) and epithelial desquamation indicate a deep level of mucosal alteration, particularly in patients with PDU and T2DM. Reduced PAS-reaction optical density in $cagA^+vacA^+$ carriers reflects more pronounced impairment of mucus formation, which worsens when PDU is combined with T2DM.

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CEREBRONENAL INTERCONNECTION ACCORDING TO THE PROOXIDANT SYSTEM OF RATS WITH ALZHEIMER'S DISEASE AND WITH CORRECTION OF THE GABA SYSTEM

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Many scientists are trying to clarify the relationship between Alzheimer's disease and renal dysfunction, which is multifactorial in nature. In particular, it is known that the physiological function of the kidney is to maintain the stability of the internal environment, including cerebrovascular blood flow, while its impairment often leads to ischemia, hypoxia and cerebrovascular diseases, which may be a significant cause of the development and evolution of cognitive decline. At the same time, there is no information in the available scientific sources about the impact of Alzheimer's disease on the condition of the kidneys.

The experiments were performed on sexually mature and elderly male rats. Alzheimer's disease was modeled with scopolamine hydrochloride, which was administered intraperitoneally at a dose of 1 mg/kg of body weight once a day for 27 days. On the 28th day, carbacetam was administered intraperitoneally at a dose of 5 mg/kg once a day for 14 days. The intensity of lipid peroxidation was assessed by the content of products that react with 2-thiobarbituric acid (TBCAP). The content of oxidative modification of proteins in homogenates was determined by the amount of their oxidative modification products by spectrophotometry.

It was established that when modeling Alzheimer's disease, we observe damage to amino groups of proteins in hippocampal neurons and endothelial cells and the mesangial matrix of non-collapsed renal glomeruli of rats of different ages with more pronounced changes in brain cells. At the same time, we see the existing relationship between damage to neurocytes and kidney cells, which is the basis for further study of pathogenetic processes and relationships in neurodegenerative processes.

Thus, in groups of rats of different ages with experimental Alzheimer's disease, an increase in the content of products of peroxide damage to lipids and proteins with more pronounced damage in hippocampal cells than in kidneys was found. At the same time, the decrease in the content of lipid and protein peroxidative damage products in the studied organs of rats with Alzheimer's disease after administration of carbacetam indicates the participation of GABA receptors in the pathogenesis of damage to hippocampal neurons and kidney cells.

Зміст

Теоретична медицина