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FEATURES OF THE SPREAD OF HELICOBACTER PYLORI INFECTION IN CHILDREN SUFFERING WITH GASTRODUODENAL PATHOLOGY

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Despite the presence of studies devoted to the study of the bacterium Helicobacter pylori, its role in the pathogenesis of diseases such as chronic gastritis, gastric and duodenal ulcers, MALT lymphoma and gastric adenocarcinoma, the question of what determines the development of one or another clinical form of gastroduodenal pathology remains open. One of the factors with which the characteristics of clinical forms of Helicobacter pylori-associated diseases are usually associated is the genetic characteristics of the microorganism, which determines its virulence.
Key words: chronic gastritis, peptic ulcer of the stomach and duodenum, Helicobacter pylori, pathogenicity factors.

Target. To study the prevalence of various genotypes of Helicobacter pylori in children with chronic pathology of the gastroduodenal zone**.**

The most studied part of the genome is the pathogenicity island, one of the chromosome segments, in which about 40 genes encoding factors that determine the virulence of Helicobacter pylori are concentrated. It is known that the most important pathogenicity factors of Helicobacter pylori, which are expected to determine the development of one or another clinical form of Helicobacter pylori infection, are the proteins CagA, VacA, IceA and BabA [2].

The CagA protein, encoded by the cytotoxin associated gene A, due to its homology to the components of the type IV secretory system of the epithelium, is integrated directly into gastric epithelial cells and leads to increased synthesis of the proinflammatory cytokine interleukin-8 [1]. Thus, human infection with cagA-positive strains of H. pylori is accompanied by a more pronounced inflammatory reaction and increases the risk of developing diseases such as peptic ulcers, gastric cancer and MALT lymphoma [3].

The vacA gene (vacuolating cytotoxin gene) encodes the formation of the vacuolating cytotoxin VacA, which causes vacuolar degeneration of epithelial cells of the gastric mucosa and their apoptosis [4]. This gene is present in the genome of all H. pylori strains and has two regions: S (signal) and m (middle), which include two allelic variants: S1/S2 and m1/m2. The vacAs1m1 genotype has a high level of cytotoxic activity and is associated with more severe diseases (peptic ulcer, gastric cancer), while the vacAs2m2 genotype does not have significant cytotoxic potential [5, 6]. Activation of the iceA gene (induced by contact with epithelium) occurs directly upon contact of Helicobacter pylori with epithelial cells. There are two allelic forms of this gene - iceA1 and iceA2. It is known that iceA1 is more often associated with peptic ulcer disease, iceA2 - with the development of chronic gastritis [8].

The membrane protein BabA (blood group antigen-binding adhesin), responsible for the adhesion of Helicobacter pylori to Lewis b-antigens of the human blood group on gastric epithelial cells [7], is encoded by the babA2 gene. In European countries, this gene is more common in patients with peptic ulcers and gastric cancer. At the same time, in a number of

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Asian countries (Taiwan, Japan), neither the presence of the babA genotype nor the combination of cagA/vacAs1 genotypes affected the outcome of infection with the bacterium H. pylori [11].

Studies devoted to studying the prevalence of various strains of H. pylori among the child population. In particular, in the work of A.A. Nizhevich et al. (2013) the prevalence of the H. pylori cagA gene in children was 46.1%, the vacAs1 genotype was 64.8%, and the vacAs2 genotype was 35.2%. A positive result when examining isolated cultures for the presence of the babA gene was obtained in 34% of children, the iceA1 and iceA2 genes - in 79.1 and 20.9% of children, respectively [9]. In 2007, according to T.V. Mishkina et al., a low prevalence of H. pylori genotypes cagA (7.91%) and vacAs1 (7.3%) was identified among children; the s2 allele of the vacuolating cytotoxin gene was found in 10.17% of children [10].

In children with gastroduodenal pathology, the frequency of the cagA gene was 34.4% of cases, the vacAs1 genotype - 60.6%, the vacAs2 genotype - 17.9%; the m1 subtype of the vacA gene was found in 45.9% of children in the study group, the VacAm2 genotype was found in 32.8% of children [12]. In our region, the distribution of Helicobacter pylori genotypes in children with gastroduodenal pathology has not previously been studied.

The study included the results of an examination of 106 children, of which 76 children whose presence of Helicobacter pylori was confirmed by polymerase chain reaction (PCR). The ratio of patients with chronic gastritis/gastroduodenitis and peptic ulcer of the stomach and/or duodenum, identified according to the results of endoscopic and morphological studies [13].

During esophagogastroduodenoscopy, all children underwent a targeted biopsy of the mucous membrane of the antrum of the stomach to verify Helicobacter pylori. Detection of Helicobacter pylori in biopsy samples was carried out using the PCR method [14].

Statistical data processing was performed using MS Excel software (Microsoft). When analyzing the data, the frequency of Helicobacter pylori genotypes was determined [15,16]. To assess the statistical significance of differences in indicators, Student's t-test was used; differences were considered statistically significant at $p < 0.05$. Helicobacter pylori was detected by PCR in all examined children (100%). The cagA gene was found in 26 (34.2%) of 76 children.

Thus, our work revealed a high percentage of cagA-negative H. pylori strains in children.

Conclusions.

1. Among the examined children, a high percentage of cagA-negative strains of Helicobacter pylori was detected.

2. Among children with gastroduodenal pathology, strains with the vacAs2m2 genotype (42.9%) dominate, which determines the low toxicogenicity of Helicobacter pylori, characterized by an average degree of virulence.

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