

VIRULENCE OF HELICOBACTER PYLORI IN PATIENTS WITH GASTROPATHY IN RHEUMATOID ARTHRITIS

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Abstract

Helicobacter pylori (H.pylori) is a gram-negative flagellar spirally twisted, microaerophilic bacterium that has urease activity, it is characterized by colonization of the gastric mucosa [1]. With H.pylori chronic gastritis, gastroduodenal ulcers, stomach tumors, MALT-lymphomas are often aggravated [2]. In addition, the prevalence of H.pylori often associated with regional characteristics, social and economic factors, age and ethnicity. H.pylori. more common in developing countries and less common in developed. There are studies related to the detection of H.pylori. bacteria, however, as a development of the development of gastroduodenal diseases, not fully understood [3]. In addition, it is known that the virulence properties and genetic characteristics of the pathogen are manifested in the form of H.pylori comorbidities [4, 5].

Target Research: assessment of the role and genotypic features of Helicobacter pylori in the formation of NSAID gastropathy in patients with rheumatoid arthritis

Materials and Research Methods

The study involved 82 patients with rheumatoid arthritis (71 (84%) women and 11 (16%) men) who were hospitalized and used NSAIDs for a long time.

The material of the study was the genomic DNA of H. pylori., isolated from the biopsy specimen of the antrum of the stomach.

Results

Our studies led to the selection and optimization of the operation of oligoprimer systems for the H. Pylori genes. The developed methodology became the basis for genotyping H. Pylori genes in RA patients with and without gastropathy, which involves preliminary molecular genetic studies to determine the frequency of occurrence of allelic variants of gene infection among apparently healthy donors and patients. Optimization of genetic and genetic studies of H. Pylori virulence genes helps to increase the efficiency and reduce the cost of research.

As a result of the molecular genetic study, there were no frequent cases among patients according to the degree of activity (> 0.05). But at the same time, in the group of patients with grade 2 RA activity, the spectrum of H. pylori genotypes was especially different, vacAm2, iceA2, and the HP gene were much more common. The cagA, vacAm1, vacAs1, vacAs1a, vacAs1b, vacAs1c, iceA1, and HP genes of H. pylori were found almost equally often in

patients with the 1st and 2nd appearance of RA activity. In patients who constantly took NSAIDs before therapy with diclofenac sodium and continued to take them in development, did not lead to an increase in the frequency of H. pylori genotypes.

Thus, as a result of the study, the following conclusion can be drawn: according to the molecular genetic study, in patients with RA 2, the pathogenic strain VacA m2, IceA 2 prevails. signs as additional markers of the onset of NSAID gastropathy in rheumatoid arthritis.

Literature

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