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THE ROLE OF HELICOBACTER PYLORI INFECTION IN THE OCCURRENCE OF CHRONIC GASTRODUODENAL PATHOLOGY

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Abstract:

Helicobacter pylori (HP) plays an important role in the development of chronic gastritis, peptic ulcer, adenocarcinoma, malt lymphoma. The review presents modern information about epidemiology, genetics and microbiology of this bacteria, factors of colonization of the gastric mucosa and the development of HP associated diseases, depending on the intraspecific diversity of strains and the presence of pathogenicity of HP, genetic predisposition, state of local and general immune factors in the protection of macrocorrhnism, changes gastrointestinal tract.

Keywords

Helicobacter pylori, pathogenesis, pathogenicity factors, congenital immunity, probiotics.

Introduction

HP is a gram -negative spiral bacterium, first isolated from the tissue of the stomach of R.Warren and B.Marshall in 1983, plays an important role in the development of a number of diseases: chronic gastritis, peptic ulcer, adenocarcinoma, malt lymphoma.

The prevalence of NR infection has significant geographical features. In most developing countries, more than 80% of the population has been infected with HP at an early age. The prevalence of HP in developed industrial countries is on average at the level of 40% and significantly lower in children and adolescents than in people of older age groups. That is, the frequency of occurrence of HP is directly related to socio-economic conditions, especially with living conditions in childhood.A rapid decrease in infection in developed countries is less likely to infect infection in childhood due to high hygienic requirements and sanitary conditions, as well as a decrease in the number of infection carriers due to antibacterial therapy. The childhood period is considered critical for the acquisition of infection of HP. So in developing countries, the level of infection of the HP is rapidly increasing among children during the first 5 years of the child's life, and then remains at a constant stable high level.In Western countries, the prevalence of HP in childhood is low, and then gradually slowly increases among adults by less than 0.5% per year [1].

Exact mechanisms for transmitting the infection of HP are unknown. HP is found only in people and some primates. Infection comes from a person with a fecal-oral or oral-oral way. This is evidenced by the detection of HP in saliva, vomiting, gastric refluxate and feces [2]. However, which of the sources is the main one is completely not clear.Obviously, infection most often occurs in early childhood from close relatives. Since the child's closest contact at the beginning of life with his mother, the problem of transmitting infection from the mother and the role of breastfeeding in this is discussed [3]. Ig to NP penetrate the placenta. Therefore, a child born from seropositive to NR of the mother can have antibodies to HP in blood [4].The risk of infection of NR during the first one and a half years of life is extremely low if the baby is on natural feeding, due to the presence of high -level IGA to NR in breast milk, which prevents the adhesion of microorganism to gastric epithelium [5]. Gastroduodenal diseases associated with HP are distinguished by a wide variety of clinical, endoscopic and morphological manifestations. The question of what determines the development of a particular form of the disease is the most difficult and has not yet been resolved. Most authors suggest the leading significance of the intraspecific diversity of strains of the HP, genetic predisposition, nature of the immune response, the duration of the disease, the degree of contamination of the NR of the gastric mucosa [7].The colonization of the HP itself is not yet a disease, but is regarded as a condition for the possible development of various lesions of the upper digestive tract, as well as, possibly, the hepatobiliary zone [8]. The ability of various strains for adhesion and invasion due to the presence of certain factors of pathogenicity, as well as the individual characteristics of the receptors of adhesion of macroorganism and will determine whether or not the disease associated or not, what will be the severity of this disease [6].It is interesting that various strains of HP can be isolated simultaneously in the same patient. This happens due to the high instability of the Genoma of the HP and the constantly occurring exchange of the genetic material between strains, as well as intra -chromosomal restructuring with the formation of new allele options [9].It is likely that the active modification of the genome and the formation of a certain phenotypic phase variation mainly with pronounced pathogenic properties is a protective reaction of microorganism to changing conditions of its existence, namely the use of antibiotics, a change in the microbial environment, and a change in acidity.

В настоящее время идентифицирован ряд факторов вирулентности Hp. Одним из ключевых факторов патогенности Hp является цитотоксичный протеин, детерминируемый геном cagA (cytotoxin associated gene), который входит в состав острова патогенности Cag-PAI. Повреждающий механизм связан с индукцией синтеза таких мощных провоспалительных цитокинов, как интерлейкин-8, интерлейкин-1 бета, фактор некроза опухоли клетками эпителия слизистой желудка и 12-перстной кишки, а также активацией нейтрофилов и мононуклеарных клеток. Another important factor in the pathogenicity of HP is VACA (vacuoolizing cytotoxin), causing vacoalization of epitheliocytes due to the formation of pores in the membrane,

activating the output of lysosomal enzymes, interaction with the elements of the cell cytoskeleton. In addition, the vacuolyizing cytotoxin is an inductor of apoptosis and an immunomodulator [10].

According to some studies, a certain role is played by: ICEA, synthesized in the contact of HP with epitheliocytes; Urea - ureaase, hydrolyzing urea and, thus, protecting the HP from the action of low pH (hydrochloric acid); Iron capture (Ferric uptake regulator - fur), riba gemolisine. The mobility of microbial cells and the chemotaxis associated with it, which contribute to the penetration of the NR into the thickness of the mucus, as well as adhesines (Baba (HOPS), OPA (HOPP)), associated with mucin, glycoproteins, glycolipids and phospholipids [20, 21].A certain role is played by the Lipopolysaccharide of the NR, the phase variations of o-specific lateral chains of which have immunological kinship with Lewis antigens of all human blood groups, which determines the phenomenon of antigenic mimicry [12]. Also play a role in the pathogenesis of infection of the protein of thermal shock of HP, which stimulate the production of interleukin-8 macrophages [13].However, on the other hand, convincing data have been accumulated that none of these factors is the predictor of the development of the disease and determines the severity of the lesion of the mucous membrane of the stomach and duodenum [14, 15].

Adhesis of HP to gastric epithelium is a critical event in the development of the infectious process. Since Helicobacter is the inhabitant of the mucous membrane, it acquired mechanisms to overcome non -specific protective factors of the gastric mucosa. Firstly, Helicobacter is able to contact the mucins of saliva and stomach [16, 17].On the one hand, it is a protective mechanism that prevents the binding of a Helicobacter directly to the surface of the epithelial cell, and on the other hand, it is a mechanism that allows the microbe to strengthen in the mucosa. And since HP has the ability to produce proteolytic enzymes that destroy mucins, this allows us to further adhere to gastric epithelium.

Probably, the oral cavity is a reservoir for HP. This is evidenced by the ability of NR to contact the mushrooms of saliva [19], ad-heat to the epithelial cells of the cheek, as well as to Fusobacterium Nucleatum and Fusobacterium Periodonticum, often detected in the dental plaque. Also, HP is able to adhere to yeast -like mushrooms of the genus Candida, especially Candida tropicalis [18].

It is obvious that not only HP itself, but also his microbial environment will determine the ability to colonize the mucosa and the manifestation of the pathogenic properties of NR.

In the mucous membrane of the stomach, with various inflammatory and erosive and ulcerative lesions, their pathogenic properties can show not only HP, but also other microorganisms belonging to the conditionally pathogenic flora (staphylococci, streptococci, mushrooms of the genus Candida, etc.). However, the nature of the severity of the pathogenicity of conditionally pathogenic microorganisms, their relationship between themselves and HP, the state of local and general immunity, against which their pathogenic potential is manifested [25], remains far from being clarified.

The pathogenesis of the development of inflammation under the action of HP is associated with the induction of the synthesis of pro-inflammatory cytokines-Interleukina-8 (IL-8), Interlayykina-6 (IL-6), Interleukina-1 beta (Il-Life), tumor necrosis factor (FNO) cells The epithelium of the gastric mucosa and duodenum with the subsequent activation of neutrophils and mononuclear cells [11]. At the same time, one of the ways to activate the secretion of data of the pro-security cytokines is the interaction of HP with thick-like receptors (TLR) of the mucous membrane of the gastrointestinal tract, playing a key role in congenital immunity [29].

Initially, it was believed that by analogy with other infectious agents of the mucous membranes the predominant protective reaction of the body for the presence of HP is the production of specific antibodies. This was confirmed by some animal experiments [22], studies on the content of antibodies to HP in breast milk [26]. However, further studies have demonstrated that the role of humoral immunity in the development of relationships between the HP and the body is sufficiently small, and the immune response develops mainly in TH1 of the mechanism [27]. Nevertheless, elimination of infection from the body does not occur.

This fact is explained by the fact that a cellular immune response is being realized through Th1, while humoral immunity is needed to combat extracellular pathogens, and the main pool of HP is represented by extracellular microorganisms. But extracellular HP, apparently, are not sufficient stimulants for the formation of antibodies.

It is possible to explain the predominant activation of the immune system by the TH1ism from the standpoint of interaction between HP with exemplary receptors. As gram -negative microorganism, HP should mainly activate TLR4. However, the Lipopolisaccharide of the NR is a weak TLR4 activator and TLR2 is more recognized, and Flagellin NR is TLR5 [28]. But unlike other gram -negative bacteria, HP does not lead to severe activation of congenital immunity reactions through interaction with TLR [23].The main role of TLR-dependent mechanisms is assumed, in particular in the reaction to the appearance of heat shock proteins [24]. Petidoglikan NR, which enters the epithelial cell in the presence of CAG PAI, interacts with another type of executive receptors-with NOD receptors. The cascade of signals launched during the activation of NOD-1 receptors may be the key in the initiation of inflammation in the mucosa [19].

The reaction of macrophages to the presence of HP is also not characteristic of a bond pathogen. It is shown that HP can inhibit phagocytosis. But the weakness of the phagocytic link lies not so much in reduced antihelicobacter activity of macrophages as impaired processes of presenting antigens of HP with dendritic cells, which is a key factor for including adaptive immunity in defense and, accordingly, the outcome of the infectious process [1].

It is interesting that, unlike other extracellular pathogens, HP induces the synthesis of Interleukin-12 (IL-12) with dendritic cells, but not IL-6 and Interlays-Na-10 (IL-10) [23]. Regulatory T cells producing IL-10 are considered one of the key factors that regulate

induced HP-drilling and allowing HP to persist in the mucous membrane of the stomach. So, it was shown that HP cannot exist in mice knocking on IL-10. However, with a lack of CD25+ regulatory T-lymphocytes, more severe forms of gastritis developed against the background of reduced microbial colonization of the gastric mucosa [24, 25].

Thus, all diseases associated with HP are not a consequence of the direct damaging effect of microorganism, but are associated with the activity of the immune response to the HP, namely the T-cell level of immunity [7, 8]. For a long time it was believed that HP is exclusively extracellular microorganism. However, the ineffectiveness of antibiotics in the schemes of eradication therapy that do not penetrate the cell in some of the patients, as well as the predominant immune response by TH1 of the type, allowed to assume the presence of intracellular forms of existence of HP. In addition, further studies using electron microscopy of biopaths of infected HP people were able to detect microorganism or parts directly inside the cells, in particular in the cells of the stomach adenocarcinoma [26, 27].There is a concept according to which HP is an integral part of human microbiocenosis for many millennia, but under the influence of adverse factors, the ability of HP to rapid mutations leads to the appearance of bacteria with pronounced pathogenic properties. Therefore, within the framework of this theory, the use of antibiotics and the conduct of, in fact, the eradication of HP is a mechanism for breeding more aggressive and resistant to antibacterial therapy of microorganisms [28].All these data are confirmed by the fact that, despite the close interest in Helicobacter infection for more than two decades, there are still many gaps in understanding the role of this microorganism in etiology and pathogenesis of human diseases. First of all, this concerns the lack of a holistic idea of the interaction of HP with other representatives of the microflora of the gastrointestinal tract and the conditions for the implementation of this bacterium of their pathogenic properties associated with a change in the microbial environment, the state of local and general immune factors of protection.

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