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Roles and Mechanisms of Action of Eradication Therapy on the Frequency of Chromosomal Aberrations and the Antioxidant System in Children with Gastroduodenal Pathology

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Abstract

It is known that various pharmacological drugs induce numerous chromosomal aberrations. The interest of doctors and scientists is focused on the question of the presence and degree of mutagenic effects of drugs used in standardized protocols of eradication therapy in children due to "rejuvenation" and the prevalence of gastroduodenal spectrum pathologies, methods of their elimination and correction, to preserve a healthy karyotype. This work aims to study the mechanisms of development of peptic ulcers, gastritis and other pathologies of the gastroduodenal spectrum associated with Helicobacter pylori, as well as the effect of eradication therapy on the frequency of chromosomal aberrations and the antioxidant system in children.

Discipline: Medicine, Gastroenterology

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1 Introduction

Several decades ago, it was assumed that peptic ulcer of the stomach and duodenum develops solely due to stress and caffeine. The opinion also prevailed that this pathology, like obesity, is caused by an incorrect lifestyle: one has only to correct habits, and everything stabilizes by itself, so doctors only recommended drinking water and not worrying about exacerbations. However, in 1982, two Australian scientists, Barry J.Marshall and J.Robin Warren, got to the truth of the causal factor in the development of peptic ulcer disease: this factor turned out to be the bacterium Helicobacter Pylori (HP) [1]. Stress and caffeine only aggravated the disease [2]. Then the scientific community did not accept the hypothesis of scientists. Maarshall had to drink a solution with bacteria, thereby infecting himself with gastritis, in order to prove the discovered relationship. It took fifteen years to recognize the rightness of the Australians. Thanks to the research of scientists, universal HP treatment protocols have already been developed, including antibiotics, gastroprotectors, antacids, M-cholinoblockers, proton pump inhibitors and histamine H-receptor blockers, and which successfully help millions of people [3].

Earlier it was believed that gastric ulcer develops from the age of 20 (the stomach – most often occurs after the age of 35, and the duodenum – from 20 to 30 years). At the age of 40 years, men suffer more often, and after 45-50 years, the ratio of the number of sick men and women is approximately the same. However, peptic ulcer disease (however, as a large-scale spectrum of gastroduodenal pathology in general, especially in chronic form) has significantly "rejuvenated". This is facilitated, according to experts, by the deterioration of environmental conditions, the irrational use of antibiotics, an increase in the number of psychoemotional stresses, poor nutrition, an increase in HP infection, etc. [4-6]. Therefore, more and more often this cohort of diseases develops in children, in whom, in particular, peptic ulcer disease is characterized by the so-called spontaneous cyclic evolution, to a greater extent than in adult patients. The meaning of this term lies in the expressed readiness for self-healing of an ulcerative defect with relapse and for an equally high readiness for another relapse. Eradication protocols for the treatment of helicobacteriosis have been adopted in children with acid-dependent diseases. At the same time, recently there is more and more data indicating the development of a number of negative reactions with this approach, which are conditionally divided into the side and toxic [7]. Adverse reactions occur as a manifestation of the main mechanism of action of a pharmacological drug, they are observed along with the therapeutic effect, and they are well studied and predictable. Specific toxic effects may have the character of mutagenic, teratogenic, embryonic and fetotoxic effects [8-10]. As for mutagenic effects, according to the literature, disorders in the structure of chromosomes not only have a negative effect on the whole organism, but also lead to the inheritance of defective genes [11]. Given the children's age of patients, this can have extremely unpleasant consequences in the long term. To date, in the structure of the pathology of the gastrointestinal tract in children, chronic gastroduodenal diseases account for 76% [12], which cannot but cause serious concern

among doctors. And it's not just the causes and prevalence of the disease: the very question of combating this problem with an eye to minimizing side effects poses a serious challenge.

It is known that various pharmacological preparations (hereinafter referred to as PP) induce numerous chromosomal aberrations (hereinafter referred to as CA). CA are divided into spontaneous and induced. Such a division is purely conditional, since the occurrence of any CA is due to certain mutagenic factors. Spontaneous aberrations are spoken of in cases where the exact trigger of their occurrence is unknown. This term was adopted in relation to the somatic cells of healthy people. Quantitative and qualitative parameters of spontaneous CA are taken as a control in the study of aberrations induced by certain factors. Chemical substances, in particular PP, can cause changes in the genetic material not only through direct interaction with the structural components of the chromosomes themselves but also through their effect on cell metabolism [13,14]. In addition to the features of the mutagenic action of substances, their effectiveness is largely determined by the stage of division at which somatic cells are located. I.e. the effectiveness of PP depends mainly on two factors: the first is the chemical properties of the compound itself, the second is the biological characteristics of cells and the organism as a whole [15,16].

A large number of new PP, and in the aggregate of mutagens as a whole, raised the problem of protecting the genotype of living beings from their destructive influence. There is very little data in the literature on the effect of eradication therapy protocols on the karyotype of children with helicobacteric lesions of the gastrointestinal tract. In this regard, the medical community has focused its attention on this problem, the main task of which is to study the effect of HP infection, schemes of eradication therapy (triple and quad therapy) on the karyotype of children and in identifying mutagenic effects, possible ways to eliminate them.

2 Pathological Disorders Underlying Peptic Ulcer and Gastritis: Developmental Factors and Genetic Markers

Normally, the stomach maintains a balance between the formation of hydrochloric acid (HCl) and mucins. Violation of this balance can lead to damage to the stomach tissue (Figure 1). There may be several reasons for the development of gastritis and ulcers: for example, the formation of mucins can be suppressed by glucocorticoids (the endogenous cause is stress, the exogenous cause is PP). Prostaglandin synthesis inhibitors (nonsteroidal anti-inflammatory drugs such as aspirin) have the same effect. On the contrary, an increase in the level of hydrochloric acid secretion may be caused by increased stimulation of the vagus nerve or an increase in the concentration of histamine or gastrin. In addition, ulcers and gastritis often develop due to infection with HP bacteria adapted to the aggressive acidic environment of the stomach [17]. Therefore, this question is polyethological. All etiological factors together potentiate each other and lead to the formation of "aggression" factors. In the end, whether or not to have peptic ulcer disease or gastritis is determined by the ratio of "protection" factors and "aggression" factors (primarily HP infection and violation of the integrity of the mucosal bicarbonate barrier, as well as a high acid-peptic factor). The causes of HCl hypersecretion are hyperplasia of parietal cells,

apparently, genetically determined vagotonia and hyperproduction of gastrin [18]. It is possible that this problem is revealed more often today: due to the negative effects of eradication therapy, including (especially in childhood).

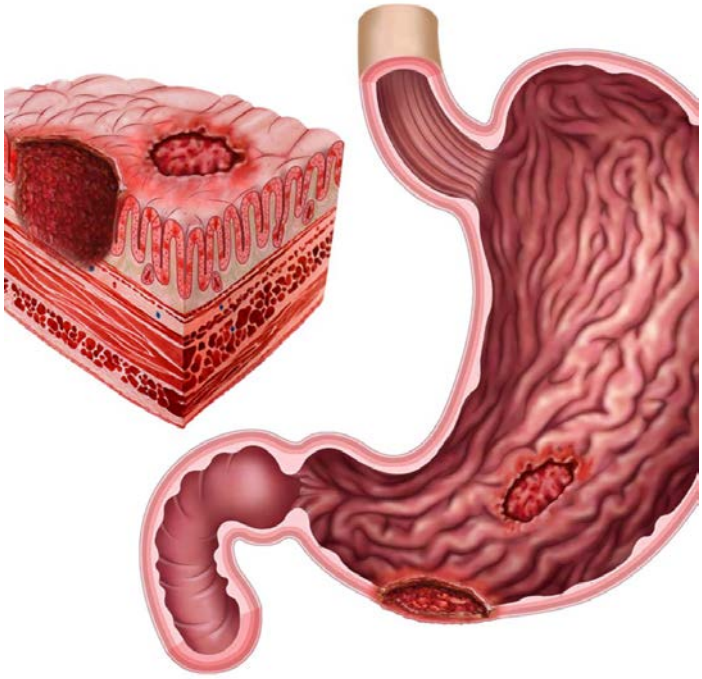

Figure 1: Damage to the gastric mucosa and parietal cells leading to gastric ulcer

It has been established that HP is detected in 90-95% of cases in patients with duodenal ulcers, and in 80% of cases in patients with gastric ulcers [19]. HP is a gram-negative anaerobic bacillus, which is found in the mucous membrane of the antrum of the stomach, sometimes detected in healthy people, without any pathological changes, but much more often (up to 95%) in patients with gastritis or peptic ulcer. Clinical symptoms were analyzed in 87 patients with chronic antral gastritis and 94 patients with chronic antral gastritis in combination with duodenal ulcers (Table 1) [20-23].

Table 1: Clinical symptoms of chronic antral gastritis and its combination with duodenal ulcer

Clinical symptom	chronic antral gastritis + duodenal ulcer		chronic antral gastritis		P
	n=94		n=87		
	abs.	%	abs.	%	
Epigastric pain	76	90,6	75	86,2	>0,05
Pain of a different localization	6	6,4	6	6,9	>0,05
Pain - free option	2	2,3	6	6,7	>0,05
Daily circadian pain	46	48,9	25	28,7	0,005
Heartburn	49	52,1	53	60,9	>0,05
Nausea	32	34,0	33	36,8	>0,05
Vomiting	18	19,1	13	14,9	>0,05
Constipation	43	45,7	19	21,8	0,0007

When it gets into the lumen of the stomach, HP finds itself in a difficult environment to live in. However, due to their urease activity, bacteria can exist in these conditions. Urease converts urea coming from the bloodstream by sweating through the capillary wall into ammonia and co2,

which neutralize (HC1) gastric juice, creating local alkalinization around the bacterial cell [23]. Ammonia acts irritatingly on the G-cells of the APUD system, increasing the secretion of gastrin and, accordingly, HC1. The morphology of bacteria provides them with active advancement, and HP, surrounded by urease and ammonia, penetrates from the lumen of the stomach into the mucus layer, where the process of advancement continues. In addition to local alkalinization, there is a decrease in the viscosity of gastric mucus around the bacteria - mucin is destroyed, and HP reaches through the protective mucosal barrier of the integumentary epithelium of the gastric mucosa. HP adheres to the integumentary-pit epithelium of the antrum of the stomach. Part of the microbes penetrates into their own plate through interepithelial contacts. Dystrophic changes occur in epithelial cells, which reduces their functional activity [24]. Intensive reproduction and colonization of HP on the antrum mucosa of the stomach leads to damage to the epithelium due to the action of phospholipases. An ulcyrogenic HP strain is isolated, which synthesizes cytotoxins that activate phospholipase. In this case, the probability of ulceration of the gastric mucosa is very high. There is a destruction of protective protein components, mucin, which opens the way for HP deep into the mucosa. Ammonia, acting on the endocrine cells of the antrum of the stomach, reduces the number of D-cells that produce somatostatin, and its concentration decreases accordingly. Gastrin release goes out of control of D-cells, which leads to hypergastrinemia, an increase in the mass of parietal cells and hyperproduction of hydrochloric acid. Thus, HP infection may be primary, and an increase in HC1 secretion may be a secondary link in the pathogenesis of gastric ulcers. An inflammatory infiltrate is formed in the submucosal layer [25]. The pathogenesis of duodenal ulcers is more complicated than gastric ulcers, but the essence is about the same (a significant difference is that HP selectively populates only the metaplastic epithelium and do not affect the normal duodenal intestinal mucosa).

In addition, HP, in the formation of ulcers plays the role of prolonged or frequently repeated psychoemotional overstrain: the coordinating function of the cerebral cortex with respect to subcortical formations and especially the hypothalamus is disrupted. There is a persistent excitation of the centers of the autonomic nervous system. Abundant pathological parasympathetic impulses from the central nervous system leads to hypersecretion of HC1 and hypermotor activity of the stomach, the release of catecholamines in synapses and adrenal medulla, which causes trophic and hemodynamic disorders in the gastric mucosa. Activation of the hypothalamic-pituitary-adrenal system causes increased production of glucocorticoids, which entails hypersecretion of gastric juice, vasospasm, catabolic effect (increased breakdown and reduced protein synthesis) [26]. All of the above causes the formation of ulcerative defects, a decrease in mucus production and a decrease in regeneration.

It has been established that genetic markers are predisposing factors of peptic ulcer disease: a high level of HC1 production - the maximum acid production of the stomach (as a result of a genetically determined increase in the mass of the lining cells and their sensitivity to gastrin), a high level of pepsinogen 1 in the blood serum - the "ulcerative pepsinogen fraction", excessive

secretion of gastrin G- cells in response to food intake, blood type I (these people have adhesive receptors to HP on the gastric mucosa), a genetically determined decrease in the production of a number of protective substances (protecting the mucosa from proteolysis), including a1-antitrypsin - serine protease inhibitor, a2-macroglobulins (make up 97% of the total macroglobulin content blood plasma - non-specific protease inhibitors and universal regulators of the immune system) [27].

3 Effect of Eradication Therapy on the Frequency of Chromosomal Aberrations and the Antioxidant System in Children with Gastroduodenal Pathology

PP represents an extensive group of chemical substances of the most diverse structure and is in most cases alien to the human body. PP can cause mutations at various levels. They can affect a very limited section of a chromosome or a single gene, in another case, an entire section of a chromosome with several genes or an entire chromosome or even the entire chromosome set may be damaged. Some PP are not inferior to ionizing radiation in terms of mutagenic effect. For example, almost all cytostatics and antimitotic antitumor drugs are able to induce gene mutations and chromosomal aberrations and cause biological effects characteristic of ionizing radiation [28]. An unspecified aspect of drug mutagenesis is the problem of assessing the genotoxic effects of combined drug effects. A clinical example of such a combined effect of drug therapy is the use of triple and square therapy for the eradication of HP in pediatric gastroenterology. In the study of Dzhagaeva et al. (2010), devoted to the study of this issue, 55 children with gastroduodenal pathology associated with HP from 7 to 17 years old participated [12]. Cytogenetic examination of the level of CA in blood lymphocytes in patients before and after treatment was performed twice according to the Moorhead method [10-11]. From the materials obtained, it follows that in children with chronic diseases of the gastroduodenal region associated with HP, the presence of infectious mutagenesis is observed, i.e. HP determines the development of an imbalance of the genetic apparatus of lymphocytes according to the criterion of accounting for HA. It follows from this that children infected with HP are carriers of "mutational cargo". The increase in the number of cytogenetic effects during eradication therapy, apparently, is a consequence of the potentiation of the actions of PP and the toxic effect of HP. After therapy, the level of CA in children increased, which is evidence of the accumulation of "active molecules" – free radicals of oxygen, nitric oxide, lipid peroxidation products, etc.

The works of other scientists also show positive results on the effect of PP from eradication therapy protocols on CA [29,30]. Filippova et al. (2010) found out that in children with diseases of the gastroenterological profile associated with HP, the level of CA is 1.3 times higher than the control, that is, there is an infectious mutagenesis [12]. Conducting eradication therapy causes an increase in the level of CA in peripheral blood lymphocytes by an average of 1.8- 2 times compared to the norm. Scientists have also found that the administration of antioxidants and ascorbic acid

can reduce undesirable side effects associated with antibiotic therapy, providing better tolerability of therapy [31].

4 Conclusion

Thus, most of the AF in eradication therapy protocols have a mutagenic effect, which, apparently, is associated with changes in the molecular processes occurring in the DNA of somatic cells under the action of these drugs. This issue deserves the close attention of scientists, whose special focus should be directed to children's patients due to the significant prevalence of gastroenterological diseases in recent years in order to prevent the development and potentiation of negative effects in the long term for each patient and for future generations.

5 Availability of Data and Material

Data can be made available by contacting the corresponding author.

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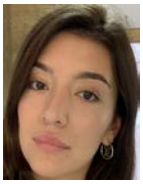

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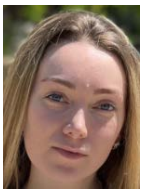

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