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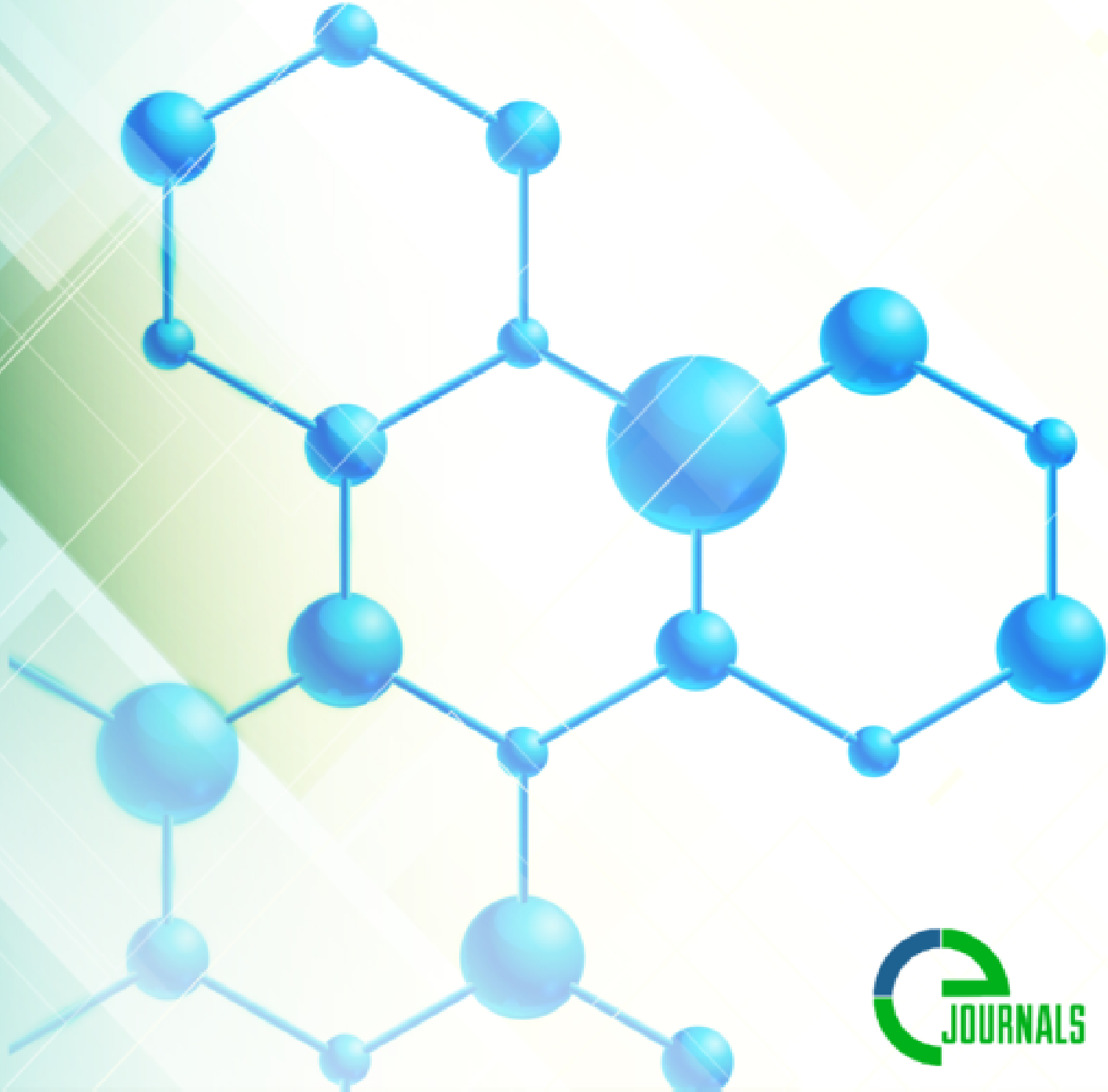

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CHRONIC GASTRITIS IN CHILDREN. ETIOLOGY AND EPIDEMIOLOGY. TREATMENT METHODS.

Alieva N.R.
Head of the Department of Hospital Pediatrics No. 1, Natural Medicine,
Doctor of Medical Sciences, Associate Professor.
Fayzieva Z.Ya
City Clinical Children's Hospital No. 5;
Republican Specialized Pediatric Research
practical medical center, emergency department doctor
dr.zulayho91@mail.ru

Introduction. Today, pathologies of the stomach and duodenum occupy a leading place among pathologies of the gastrointestinal tract in children (1,2). Chronic lesions of the stomach account for 70-75% of pathologies of the gastrointestinal tract. Currently, on average, more than half of people worldwide suffer from chronic gastritis. Helicobacter pylori, an infection in childhood, is the main cause of chronic gastritis, the microbial origin of which is the key to understanding the strange epidemiology and course of the disease. Prolonged and aggressive inflammation during gastritis over time (years and decades) leads to destruction (atrophic gastritis) of the gastric mucosa. Currently, the importance of gastritis as a serious disease is largely underestimated, although the role of gastritis in the pathogenesis of gastric ulcers and gastric cancer is obvious [3].

Keywords: Gastritis, Helicobacter pylori, children, peptic ulcer.

Chronic gastritis is a chronic, recurrent, progressive inflammatory-dystrophic lesion of the mucous membrane of the stomach and duodenum, accompanied by disruption of the physiological processes of regeneration and proliferation, as well as various secretory and motor-evacuation disorders [15,16].

Chronic gastritis has been known and studied since the first decades of the 20th century, but more attention was paid to it only in 1982, after the discovery of Helicobacter pylori by Warren and Marshall [17].

Epidemiology. Chronic gastritis in children remains a common disease, including in developed countries.

Gastritis caused by H. pylori is mainly acquired in early childhood, and the course of gastritis is a harmless and asymptomatic simple chronic mononuclear ("superficial") inflammation [7, 8]. Severe symptomatic consequences of chronic gastritis appear only in the last decades of life and, as a rule, occur in patients with late stages of damage.

Since the discovery of H. pylori, it has become apparent that understanding the epidemiology of H. pylori infection is key to understanding the epidemiology of chronic gastritis as well as its consequences.

The most important background factors in the epidemiology of chronic gastritis, as well as in the transmission of H. pylori infection throughout the world, are socio-economic factors and environmental hygiene [12, 13, 14]. Socioeconomic status during childhood, environmental and family health, family density, food preparation habits, etc. are likely factors that determine the likelihood of contracting H. pylori infection during childhood. Improving these conditions reduces the likelihood of infection or further transmission of infection, most likely through the gastro-oral route. Reducing the rate of infection in childhood at the family level will inevitably lead over time to a decrease in the prevalence of both H. pylori gastritis and its consequences, as well as in

the general population on average.

Etiology. The most common cause of gastritis in children is dietary errors. Factors predisposing to the development of gastroduodenal diseases in children, especially from the first years of life, may be an early transition to artificial feeding, inadequate and irregular nutrition, abuse of spicy and hot foods, and poor chewing of food. According to many authors, in children aged 1.5 to 7 years with a burdened antenatal history, the risk of early development of pathology of the digestive organs increases 4 times. In addition to the nutritional aspect, pathogenic microorganisms play an important role in the etiology of gastritis. Most often, the cause of the disease is bacteria that enter the child's stomach with poor-quality food (food poisoning). A similar effect occurs when using aggressive medications, alcohol or drugs.

An important endogenous cause of gastritis in children is the bacterium Helicobacter Pylori. The pathogen normally lives in the pyloric part of the stomach, and if there are appropriate conditions for its reproduction, it affects the entire surface of the mucous membrane of the organ. The bacterium secretes aggressive toxins that irritate the inner layer of the stomach and provoke inadequate secretion of hydrochloric acid. The result of this phenomenon is the development of gastritis.

Provoking factors are also considered:

- chronic diseases of other gastrointestinal organs in the acute stage (cholecystitis, pancreatitis);
- hereditary predisposition;
- irregular eating;
- consumption of large amounts of fast food, snacks (chips, crackers), carbonated drinks;
- seasonal weakening of immune defense;
- uncontrolled use of medications (antipyretics, antibiotics, hormonal agents).

Cases of gastritis in preschool children are not uncommon. Children often complain of abdominal pain. More often the pain is localized in the epigastric region and right hypochondrium. Also, diffuse abdominal pain is common, there is a decrease in appetite, an aversion to a number of foods (porridge, dairy dishes, etc.), nausea and vomiting, which usually begins 2-3 hours after eating, and heartburn may occur. , sour belching, a rise in temperature to low-grade levels is also not excluded. The pain intensifies with physical activity (running, jumping, fast walking, etc.). About half of patients experience constipation.Most patients experience increased excitability, irritability, and emotional instability.

The clinical picture of gastritis will manifest itself depending on the localization and extent of the inflammatory process [18, 19, 20].

In case of damage to the antrum of the stomach and with antroduodenitis, the disease proceeds in an ulcer-like manner.

Three syndromes are characteristic: pain, dyspeptic, asthenovegetative.

The pain syndrome is manifested by intense abdominal pain, which:

- localized in the epigastric region and pyloroduodenal zone;
- may occur 1.5-2 hours after eating or on an empty stomach, sometimes at night;
- decrease or stop after eating;
- may be accompanied by heartburn; sometimes sour belching;
- occasional vomiting, which brings relief.

Asthenovegetative syndrome in patients is manifested by irritability, headaches, sweating, drowsiness, fluctuations in blood pressure, and sleep disturbances.

Dyspeptic disorders (heartburn, sour belching) are usually associated with insufficiency of the cardiac sphincter of the esophagus.

When examining and examining patients, the following is determined:

- tongue coated with white coating;
- pain on palpation of the abdomen in the epigastric region and/or pyloroduodenal area;
- A positive Mendelian symptom is noted, which distinguishes gastroduodenitis from isolated gastritis and duodenitis;
- preserved appetite;
- stool will be prone to constipation;
- changes in the mucous membrane of the antrum of the stomach and duodenum in the form of inflammation, subatrophy and/or hyperplasia of the mucosa, erosive lesions (during endoscopy);
- preserved or increased function of acid and enzyme formation;
- discoordination of the secretory and motor functions of the stomach and duodenum.

With inflammation of the fundus of the stomach, inflammatory activity, as a rule, occurs in a gastritis-like manner.

Pain syndrome is characterized by the fact that pain:

- low-intensity, dull, early (occur after eating, especially after heavy, fried and fatty foods);
- localized in the epigastric region and the navel area;
- go away on their own within 1-1.5 hours. Dyspeptic manifestations - a feeling of heaviness, fullness in the epigastrium after eating, nausea, sometimes belching air; Occasional vomiting of eaten food, which brings relief.

Asthenovegetative syndrome - patients experience weakness, lethargy, fatigue, and general malaise.

When examining and examining patients, the following is revealed:

- abdominal pain on palpation in the upper and middle third of the space between the xiphoid process and the navel;
- decreased and selective appetite;
- flatulence and loose stools;
- inflammatory, atrophic, subatrophic changes, focal atrophy, and often multiple erosions of the middle third of the gastric mucosa are detected (during endoscopy);
- reduction of acid and enzyme formation;
- violation of the motor function of the stomach in the form of atony.

In addition to the main clinical forms of chronic gastritis/gastroduodenitis, the development of many atypical and asymptomatic forms is possible. Chronic gastritis/ gastroduodenitis also occurs latently, and in these cases, morphological changes and clinical symptoms may not coincide.

In the stage of clinical remission there is no pain or dyspeptic symptoms, but instrumental (endoscopic, ultrasound) studies reveal the presence of changes in the mucous membrane of the stomach and duodenum. Exacerbations more often occur in spring and autumn and are associated with stressful situations, overload at school, and eating disorders [20].

Treatment. The first stage of treatment is aimed at reducing the effect of aggression factors (suppression of the acid-peptic factor, eradication of H. pylori, relief of hypermotility and dysfunction of the central and autonomic nervous system) [21]. The second stage of treatment is the restoration of resistance of the mucous membrane of the stomach and duodenum. The third stage is restorative treatment (mostly non-drug) to normalize the functional and morphological state of the cells of the mucous membrane of the stomach and duodenum [22]. Anti-Helicobacter therapy is considered the main standard of

treatment for Helicobacter-associated acid-related diseases, as reflected in international recommendations (Maastricht Agreements 1-4, 1996, 2000, 2005 and 2010, respectively). Unfortunately, in recent years, an increase in the number of patients receiving anti-Helicobacter therapy has led to an increase in the number of antibiotic-resistant strains of H. pylori, which is the main reason for the ineffectiveness of the treatment. Ways to overcome resistance are different, these include lengthening the duration of treatment, combining drugs taking into account the synergism of their anti-Helicobacter activity, and searching for new effective drugs [23, 24]. Non-drug treatment includes adherence to the treatment regimen and diet. So, in case of exacerbation of gastritis, the diet should be gentle (table No. 1 according to Pevzner), and if the child receives colloidal bismuth tripotassium citrate, then a dairy-free diet is indicated (table No.4), as in the case of intestinal pathology. You should stop taking medications that have an adverse effect on the gastric mucosa (for example, non-steroidal anti-inflammatory drugs) [22, 25].

Conclusion.

The diagnosis of chronic gastritis can only be established after a morphological examination of biopsies of the gastric mucosa. Effective eradication treatment of H. Pylori guarantees only a relatively favorable prognosis for chronic inflammation of the gastric mucosa. Repeated reinfection with H. pylori occurs in 1-1.5% (provided that there are no carriers of the microorganism in the child's environment; in this case, reinfection occurs in 15-30%). After eradication of H. pylori, the inflammatory reaction of the gastric mucosa disappears within 2 to 6 months. In the absence of symptoms, the disease does not require additional treatment [22, 26]. Thus, unsolved problems in the treatment of chronic gastritis in childhood remain the lack of organization of proper nutrition for children and adolescents at home and at school, stressful situations, the need for constant use of drugs that have an aggressive effect on the mucous membrane of the stomach and duodenum, reflux gastritis, resistance of H. pylori to anti-Helicobacter drugs, carriage of Helicobacter by members of the child's family, especially highly virulent strains, poor patient compliance with doctor's prescriptions for eradication or self-medication, genetic predisposition of the gastric mucosa to alteration, bad habits, concomitant diseases of the digestive system. Each of these factors is multifaceted and requires detailed study at both the individual and population levels; joint participation in scientific work of pediatricians, gastroenterologists, morphologists, hygienists, microbiologists, clinical pharmacologists and other specialists.

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