

## IMPROVING THE QUALITY OF LIFE OF PATIENTS WITH CHRONIC GASTRITIS

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**Abstract:** Helicobacter inhabits the gastric mucosa, causing inflammation and damage. The source of infection is an infected person. It is believed that about 80% of cases of HG are associated with Helicobacter pylori. Chronic gastritis is an inflammatory disease caused by gastric complications caused by infections (most often Helicobacter pylori), autoimmune processes, long-term use of certain medications, as well as bad habits and poor nutrition. The most common cause is damage to the gastric mucosa by the bacterium Helicobacter pylori.

**Key words:** prognosis, chronic gastritis, risk factors, early diagnosis, prevention.

**Relevance:** Chronic gastritis is a group of chronic diseases that are morphologically characterized by persistent inflammatory infiltrate and impaired cellular renewal with the development of intestinal metaplasia, atrophy and epithelial dysplasia in the gastric mucosa. Gastritis is an inflammatory disease of the gastric mucosa. Acute and chronic gastritis are distinguished. [1]. Chronic gastritis, despite the apparent simplicity of the disease, continues to create difficulties for clinicians and practicing doctors both in diagnosis and treatment. These difficulties primarily concern the differentiation of gastritis from functional dyspepsia, the type of gastritis itself, as well as the tactics of patient management and prevention of relapses. This is often due to the difficulties of diagnosis, as well as the multifactorial nature of the causes of the disease. Long-term gastritis with relapses reduces patients' adherence to the doctor's recommendations, which exacerbates the difficulties in treatment [2]. Chronic gastritis is a group of chronic diseases that are morphologically characterized by the presence of inflammatory and dystrophic processes in the gastric mucosa, its progressive atrophy, functional and structural restructuring. Gastritis may have various clinical manifestations or be asymptomatic [3]. Therefore, chronic gastritis is primarily a morphological concept. Inflammation in chronic gastritis is limited to the gastric mucosa. The result of chronic inflammation is a decrease in the mass of functionally active glandular tissue, that is, the development of atrophic gastritis [4,5]. The most common cause of chronic gastritis is Helicobacter pylori infection, which is associated with the high prevalence of this microorganism in the population. Thus, in the course of an epidemiological study conducted in 2006 using a urea breath test with C13-

labeled urea, it was shown that the prevalence of HP in Moscow was 60.7% [5]. Despite numerous studies devoted to chronic gastritis, information on the prevalence of chronic gastritis in different countries of the world is not clear [6,7]. There is a point of view that salt is capable of potentiating the processes of gastric carcinogenesis, mainly in patients with *Helicobacter* gastritis, but it is not fully confirmed by the results of existing studies. One of the reasons for the gradual decrease in the incidence of stomach cancer in many populations over the past 50 years is considered to be the widespread use of household refrigerators, which has significantly reduced the need to use salt as a food preservative [5,7]. Gastritis changes are reactions of the gastric mucosa in response to various endogenous and exogenous pathogenic factors. The main changes that make up the morphological picture of chronic gastritis include inflammation, atrophy, and disruption of cellular renewal, including metaplasia and dysplasia. The *Helicobacter pylori* bacterium, colonizing the gastric mucosa, is the etiologic factor of gastritis. The establishment of the etiologic significance of *H. pylori* made chronic gastritis a clearly defined and clinically significant nosological entity - a disease with a known cause, stages of pathogenetic development, a certain prognosis and, finally, determined the possibilities of etiotropic treatment [1,7,9].

The main functions of the stomach are:

1. Storage of food.

2. Partial digestion. Digestion of proteins and cellulose begins in the stomach. Under the influence of hydrochloric acid, proteins are denatured and plant cellulose swells. Under the influence of pepsin and gastrin, proteins are broken down into peptides and amino acids. Pepsin is produced by the main cells in the form of pepsinogen, which, under the influence of hydrochloric acid, passes into an active form.

3. Partial absorption. Some medications (acetylsalicylic acid, barbiturates) and alcohol can be absorbed in the stomach.

4. Conducting food to the intestines. Food stays in the stomach for an average of 3-10 hours. Moreover, liquid is evacuated immediately, fatty foods are retained the longest. The vagus nerves increase muscle activity, and the sympathetic ones decrease it.

5. The bactericidal function is realized due to the action of hydrochloric acid. Therefore, dysbacteriosis is an invariable companion of hypoacid conditions, as well as long-term use of proton pump inhibitors (omeprazole).

6. Hematopoietic function (synthesis of Castle's intrinsic factor).

Due to the marked decrease in the functional activity of the stomach in atrophic gastritis, the leading syndrome is dyspepsia, in contrast to hyperacid gastritis, where pain syndrome predominates [8,9,10]. The appearance and development of chronic gastritis is determined by the impact of many factors on the stomach tissue. The main

external etiological factors contributing to the development of chronic hepatitis are: The most significant are infection of the stomach with *Helicobacter pylori* and, to a lesser extent, other bacteria or fungi; nutritional disorders; bad habits: alcoholism and smoking, long-term use of drugs that irritate the gastric mucosa; exposure of the mucosa to radiation and chemicals; parasitic infestations; chronic stress. Internal factors that contribute to the development of chronic gastritis are: genetic predisposition; duodenogastric reflux; autoimmune processes damaging stomach cells; endogenous intoxications; hypoxemia; chronic infectious diseases; metabolic disorders; endocrine dysfunctions; vitamin deficiency; reflex effects on the stomach from other affected organs [9].

**Purpose of the study:** The objective of this study is to develop a program for early diagnosis of *Helicobacter gastritis* in a polyclinic setting and to establish clinical manifestations of *Helicobacter gastritis*. o study the etiology and dynamics of prevalence in chronic gastritis of *Helicobacter pylori* etiology. To determine the importance of prevention and the impact of chronic gastritis on health. Among the diseases of the digestive system, chronic gastritis (CG) is the most common. Having a tendency to frequent relapses, involving other organs of the digestive system in the pathological process, leading to complications that threaten the lives of patients, these diseases quite often become the cause of temporary and permanent disability, especially in young people. This is what determines the need to develop and implement scientifically based methods of primary prevention in practical healthcare, since no advances in diagnostics and treatment will lead to a decrease in the incidence of chronic hepatitis in the population. The identification of individual unfavorable health risk factors that influence the occurrence of the disease, and risk groups among the population, represents significant opportunities for the development of measures for the primary prevention of chronic hepatitis [10,12]. Existing methods of diagnosing chronic gastritis are more often used in hospital settings and are quite invasive. A wider introduction of non-invasive research methods and screening diagnostic tests is necessary here [11]. Determination of the titer of antibodies to gastric parietal cells (APC) and Castle's intrinsic factor allows us to confirm the immune nature of gastritis, but recent studies have shown that these markers may be negative in some patients [12,13].

**Materials and methods of the study:** the object of the study were 35-year-old residents of the city of Tashkent. The total sample size was 199 people, of which 100 were patients (the main group) and 99 individuals not burdened with chronic hepatitis (the control group). Primary social and hygienic information about the sample, data on accumulated morbidity and factorial characteristics, were obtained by copying information on chronic diseases from outpatient cards and interviewing research subjects using specially developed questionnaires. The questionnaires include

characteristics of 90 variables of conditions and lifestyle for the main group and 70 for the control group. Considering the fact that chronic disease is often the cause of changes in the patient's conditions and lifestyle, the questionnaire included those characteristics of factors that were inherent in the person before the initial registration of the diagnosis of chronic hepatitis.

**Results:** Subsequently, detailed analytical work was carried out according to specially developed programs. In each case of reliable connection between unfavorable risk factors and disease ( $p < 0.06$ ), determination coefficients and relative risk values were determined as a central indicator in analytical epidemiology, independent of the incidence rate in the reference group. Among patients in both the main and control groups, women predominated (64.2 and 68%, respectively). The analysis revealed a total of 38 common factors that reliably influence the occurrence of chronic gastritis, of which 11 are unfavorable and 20 are favorable factors that increase or decrease the likelihood of disease occurrence. Since for chronic gastritis the female gender itself is a risk factor, the information content of common unfavorable and favorable risk factors for chronic gastritis is carried out by gender. An analysis of the health status of first-degree relatives showed that 77.3% of patients in the main group and 84% of patients in the control group had parents who suffered from gastrointestinal diseases. Chronic gastritis and chronic gastroduodenitis predominated in the structure of diseases among relatives. Early pain was recorded significantly more often in the control group ( $61.2 \pm 7.8\%$ ). Patients with chronic gastritis of *Helicobacter pylori* etiology, on the contrary, more often complained of episodic ( $65.7 \pm 4.3\%$ ), moderate ( $72.7 \pm 3.9\%$ ), monotonous ( $81.3 \pm 4.1\%$ ) pain localized in the epigastrium ( $95.2 \pm 1.2\%$ ). Among the manifestations of gastric dyspepsia in the control group, nausea ( $92.4 \pm 4.1\%$ ) and constipation ( $39.0 \pm 8.0\%$ ) predominated. Manifestations of asthenovegetative syndrome increased with the age of patients, which could be associated with an increase in the duration of the disease. Thus, in patients aged 40 years, fatigue, irritability, and weakness were more common. In addition, their asthenovegetative manifestations were varied and included headache, dizziness, sweating, and insomnia.

**Conclusions:** Among the conditionally controlled factors influencing the occurrence of chronic gastritis, the level of hereditary predisposition is of significant importance. There is information about the role of constant depression, night shifts, heavy physical labor without adequate rest; dry, cold or hot food, alcohol abuse in the development of chronic gastritis. The prevalence of chronic gastritis depends more on the combination of unfavorable factors than on health-promoting factors, which is confirmed by the following calculations. [10,11,13]. Some authors also point to the great importance of the consumption of genetically modified foods, smoking, the use of preservatives, flavor enhancers, sugar substitutes and emulsifiers, the presence of infectious processes in the liver, pancreas and other organs in reducing the protective

functions of the gastric mucosa during the formation of chronic gastritis. Thus, it is still unclear what factors influence the development of chronic gastritis in the population [6,7,13]. A number of authors point to the presence of a hereditary predisposition; they believe that the share of influence of genetic factors fluctuates within 70%. According to our data, for men, the presence of digestive diseases in the father increases the probability by 1.5 times, and their presence in the mother - by 1.4 times. For women, the presence of digestive diseases in the father increases the probability of developing PU by 2 times, and if they are present in the mother - accordingly, CG by 1.3 times [10,14,15]. Thus, our research has shown that in a polyclinic setting, the following program-algorithm can be used to diagnose chronic gastritis and its etiology;

- study of hereditary history and disease history; - detailed analysis of clinical manifestations; - use of non-invasive diagnostic methods: breath test and analysis of IgG antibodies to H.

The article actualizes the problem of early diagnosis of chronic gastritis and demonstrates the importance of practical application of currently existing non-invasive methods for diagnosing gastric diseases. Endoscopic and morphological examination can be used in cases where it is necessary to exclude peptic ulcer disease, as well as in cases of sluggish gastritis with frequent exacerbations.

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