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Clinical and Laboratory Diagnostics and Modern Treatment Strategies for Gastritis

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Abstract: This scientific article provides a detailed analysis of the etiopathogenesis of acute and chronic gastritis, modern classification systems, contemporary laboratory and instrumental diagnostic methods, and treatment regimens based on international protocols. The article places special emphasis on invasive and non-invasive methods for detecting Helicobacter pylori infection, assessing gastric acidity, and analyzing biomarkers. Additionally, it covers eradication therapy based on the Maastricht VI consensus, new generations of antisecretory drugs, and strategies for preventing neoplasia.

Keywords: Chronic Gastritis, Helicobacter Pylori, Kyoto Consensus, OLGA System, Eradication, Potassium-Competitive Acid Blocker, Gastropanel

Introduction

Chronic gastritis is a heterogeneous group of diseases characterized by long-term, immune-inflammatory, and dystrophic changes in the gastric mucosa, which culminate in impaired regeneration processes, cellular atrophy, and the development of metaplasia and dysplasia [1]. According to epidemiological data, more than 50% of the world's population suffers from chronic gastritis. The significance of the disease is determined not only by its high prevalence but also by its position as the starting point of the "Correa's Cascade" Normal GM → Chronic Gastritis → Atrophy → Intestinal Metaplasia → Dysplasia → Adenocarcinoma, which is considered the basis for the development of gastric adenocarcinoma. International gastroenterological studies from 2020–2026 indicate that the correct laboratory and endoscopic diagnosis of gastritis at an early stage reduces the risk of stomach cancer by 60–70%.

Etiology and Pathogenesis

According to modern concepts, the primary factor in the development of gastritis is Helicobacter pylori infection in 80-85% of cases. Nonsteroidal anti-inflammatory drugs and autoimmune processes are the second most common causes [2].

1. Pathogenesis of H. pylori-induced gastritis

In the acidic environment of the stomach, H. pylori breaks down urea into ammonium and carbon dioxide with the help of the enzyme urease. Ammonium neutralizes the acid, creating a protective shell around the bacterium. The bacterium's virulence factors, such as CagA cytotoxin-associated gene A and VacA vacuolating cytotoxin A, have a cytotoxic effect on the gastric epithelial cells, induce the synthesis of interleukin-8, and cause the infiltration of neutrophils and macrophages. This leads to chronic active gastritis.

2. Pathogenesis of autoimmune gastritis

In this form, the body produces autoantibodies against the parietal cells of the gastric fundus and

body, as well as against the Intrinsic Factor. The destruction of parietal cells leads to a sharp decrease in hydrochloric acid production and impaired vitamin B12 absorption, resulting in pernicious anemia [3].

Modern International Classification Systems

In addition to the traditional Sydney classification, two main systems are currently used in international scientific and clinical practice:

1. Kyoto Classification of Gastritis

Based on the etiological principle, this classification fundamentally regrouped gastritis as follows:

1. H. pylori-induced gastritis (regarded as a separate infectious disease.
2. Drug-induced gastritis, e.g., caused by NSAIDs.
3. Autoimmune gastritis.
4. Specific gastritis lymphocytic, eosinophilic, granulomatous.

2. OLGA System

This system assesses the precancerous condition based on the degree of atrophy in biopsy samples taken from 5 points in the antrum and gastric body. Based on the extent and location of the atrophy, the patient is assigned Stage I, II, III, or IV. Patients in Stages III and IV are considered a high-risk group for the development of gastric cancer and are placed under dispensary follow-up [4].

Clinical Presentation

Chronic gastritis can be asymptomatic for a long period. When symptoms do appear, they are mainly divided into the following groups:

1. Gastric Dyspepsia Syndrome: Post-meal heaviness in the epigastric region, a feeling of fullness, early satiety, belching, and nausea.
2. Epigastric Pain Syndrome: Aching or burning pain in the epigastrium related to meals (often on an empty stomach or 1.5-2 hours after eating).
3. Systemic Disorders (in Autoimmune Gastritis): Anemia resulting from B{12} deficiency, a burning sensation of the tongue, Hunter's glossitis, paresthesia, and general weakness [5].

Clinical, Laboratory, and Instrumental Diagnostics

According to modern international standards, a diagnosis of gastritis cannot be based solely on clinical symptoms. It requires laboratory and endoscopic confirmation.

1. Non-invasive Laboratory Diagnostics

The Gastropanel complex, also known as a "serological biopsy," assesses the functional and structural state of the mucous membrane through a blood test:

Pepsinogen I and Pepsinogen II: PG I is synthesized in the body of the stomach. Its decrease and a low PG I/PG II ratio <3 indicate atrophy of the gastric body [6].

Gastrin-17 G-17: Produced by G-cells in the antrum. When acidity decreases, the level of G-17 increases as a compensatory mechanism. In cases of antral atrophy, G-17 levels approach zero.

H. pylori antibodies: Indicate infection.

2. Methods for Diagnosing H. pylori Infection

According to the Maastricht VI recommendations, diagnostic methods are categorized as follows [7]:

Table 1. Comparative Characteristics of Invasive and Non-Invasive Diagnostic Methods for *Helicobacter pylori* Detection

Method type	Test title	Sensitivity and Specificity	Features
Non-invasive	^{13}C-urea breath test (^{13}C-UBT)	>95\%	The gold standard for verifying eradication efficacy
Non-invasive	Fecal H. pylori antigen detection (EIA)	>93\%	Convenient and affordable for screening and monitoring
invasive	Rapid Urealase Test (RUT)	>90\%	It is performed with a biopsy sample obtained during endoscopy.
invasive	Histological examination (Modified Giemsa)	>95\%	Simultaneously assesses atrophy and metaplasia.
invasive	PCR (Molecular) and Sequencing	>98\%	Detects resistance to clarithromycin

Important Note: It is essential to stop taking proton pump inhibitors (PPIs) at least 2 weeks before, and antibiotics and bismuth preparations at least 4 weeks before, any H. pylori test. Otherwise, the analysis may yield a false-negative result.

3. Endoscopic and Histological Examination

Modern EHE should be performed in high definition with NBI mode, which visualizes mucosal capillaries, or with chromoendoscopy for enhanced visualization. A biopsy is taken from at least 5 points according to the Sydney Protocol: 2 from the antrum, 2 from the body of the stomach, and 1 from the incisura angularis (gastric angle) [8].

Modern Treatment Methods

The treatment of gastritis is based on its etiology, histological findings, and acid secretion function.

1. H. pylori Eradication Therapy (Maastricht VI Standard)

Due to the increasing level of antibiotic resistance, treatment regimens are selected based on regional resistance patterns. As resistance to clarithromycin is high in Uzbekistan and the Central Asian region, bismuth-containing quadruple therapy is primarily recommended as the first-line treatment.

First-Line Therapy: Bismuth Quadruple Therapy (Duration: 10-14 days)

1. PPI Proton Pump Inhibitor: Esomeprazole 20 mg or Rabeprazole 20 mg twice daily, 30 minutes before meals.
2. Bismuth tripotassium dicitrate 120 mg four times daily or 240 mg twice daily protects the mucous membrane and breaks down the bacteria.
3. Metronidazole 500 mg, 3-4 times daily.
4. Tetracycline 500 mg, four times daily, or Amoxicillin 1000 mg twice daily.

Second-Line Therapy for relapse after non-quadruple therapy: Levofloxacin-based regimen

• PPI + Amoxicillin 1000 mg + Levofloxacin 500 mg + a Bismuth preparation.

2. New-Generation Antisecretory Therapy: P-CABs

In recent years, the biggest innovation in gastroenterology has been the introduction of P-CABs (Potassium-Competitive Acid Blockers). This class includes Vonoprazan.

• Advantage: Unlike traditional PPI drugs, Vonoprazan does not require an acidic environment for activation. It rapidly raises the gastric pH to >4 from the first dose and maintains it at a stable level

for 24 hours. This dramatically increases the efficacy of antibiotics.

3. Mucoprotectors and Gastroprotectors

These are used to accelerate mucosal regeneration after eradication therapy or in cases of gastritis with normal acidity:

Rebamipide: Stimulates the synthesis of prostaglandins in the mucous membrane, increases the amount of glycoproteins, and restores the epithelial barrier function. The course of treatment is 2-4 weeks.

Solphatran/Sucralfate: Forms a protective film over erosive processes.

4. Treatment of Autoimmune Gastritis

There is no specific cure for autoimmune gastritis. The main focus is on managing its complications:

To treat vitamin B12 deficiency, Cyanocobalamin is administered parenterally for life.

If iron-deficiency anemia is observed, iron supplements are prescribed.

According to the MAPS II Management of epithelial precancerous conditions and lesions in the stomach International guidelines:

Patients with mild atrophy confined to either the antrum or the body of the stomach do not require endoscopic monitoring.

Patients with severe atrophy or intestinal metaplasia in both the antrum and the body of the stomach must undergo high-definition EHE with biopsy every 3 years.

Results and Discussion

The analysis of current scientific literature demonstrates that gastritis remains one of the most prevalent gastrointestinal disorders worldwide, with a multifactorial etiology involving infectious, autoimmune, chemical, and environmental factors. Among these, *Helicobacter pylori* infection continues to be recognized as the leading cause of both acute and chronic gastritis. The reviewed evidence confirms that persistent colonization of the gastric mucosa by *H. pylori* induces chronic inflammation, leading to progressive mucosal damage, glandular atrophy, intestinal metaplasia, and, in some cases, gastric adenocarcinoma [9].

Modern classification systems, particularly the Updated Sydney System and the Kyoto Global Consensus, have significantly improved the standardization of gastritis diagnosis by integrating endoscopic, histological, and etiological criteria. These approaches facilitate more accurate disease staging and risk stratification for gastric neoplasia. The findings indicate that histopathological evaluation remains the gold standard for assessing the severity of mucosal inflammation and atrophic changes [10].

Diagnostic advances have substantially enhanced the detection and monitoring of gastritis. Invasive methods, including endoscopic biopsy with histological examination, rapid urease testing, bacterial culture, and molecular techniques, provide high diagnostic accuracy and allow direct assessment of gastric mucosal alterations. However, non-invasive diagnostic tools such as the urea breath test, stool antigen test, and serological biomarkers have gained increasing clinical importance due to their accessibility, safety, and effectiveness in detecting *H. pylori* infection and evaluating treatment outcomes [11].

The assessment of gastric acidity and biomarkers, including pepsinogen I/II ratio, gastrin-17, and anti-parietal cell antibodies, has emerged as an important component of contemporary gastritis management. These biomarkers contribute to the identification of atrophic gastritis, autoimmune gastritis, and patients at elevated risk for gastric cancer. Their integration into diagnostic algorithms supports earlier intervention and improved long-term prognosis [12].

The reviewed treatment strategies emphasize the importance of eradication therapy according to

the Maastricht VI Consensus recommendations. Evidence suggests that optimized quadruple therapy regimens achieve higher eradication rates compared with traditional triple therapy, particularly in regions with increasing antibiotic resistance [13]. The use of bismuth-containing regimens and susceptibility-guided treatment approaches further enhances therapeutic success.

Recent developments in antisecretory therapy have introduced more potent and longer-acting acid-suppressive agents. Potassium-competitive acid blockers (P-CABs), such as vonoprazan, demonstrate superior acid control and may improve eradication outcomes when compared with conventional proton pump inhibitors. These agents represent a promising advancement in the management of gastritis and related acid-dependent disorders [14].

Furthermore, preventive strategies aimed at reducing the progression from chronic gastritis to gastric neoplasia have become increasingly significant. Early detection and eradication of *H. pylori*, regular surveillance of patients with atrophic gastritis or intestinal metaplasia, and the application of biomarker-based risk assessment models contribute to effective cancer prevention. The reviewed evidence highlights that a comprehensive diagnostic and therapeutic approach can significantly reduce disease burden and improve patient outcomes [15].

Overall, the findings underscore the necessity of combining modern diagnostic technologies, evidence-based eradication protocols, and individualized patient management strategies to optimize the diagnosis, treatment, and prevention of gastritis and its complications.

Conclusion

Chronic gastritis is not a simple dietary disorder but a pathology with a deep genetic and microbiological basis. Modern diagnostics should rely on "Gastropanel" serological screening and high-definition endoscopy. In terms of treatment, adherence to the international Maastricht VI standards for H. pylori eradication and the use of innovative molecules like Vonoprazan in difficult-to-treat cases are the most effective measures for preventing stomach malignancies.

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