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CLINICAL COURSE AND THERAPEUTIC STRATEGIES OF CHRONIC GASTRITIS

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Abstract: Chronic gastritis is a persistent inflammatory disease of the gastric mucosa that develops gradually and can progress silently over time. Its clinical course is often nonspecific, marked by symptoms such as epigastric discomfort, early satiety, nausea, and bloating, which are frequently overlooked. The condition is most commonly associated with *Helicobacter pylori* infection, autoimmune reactions, or prolonged NSAID use. If left untreated, chronic gastritis can lead to atrophic changes, anemia, and even gastric carcinoma. Accurate diagnosis through endoscopy and histology, along with etiologically guided therapy, is essential for effective management. Treatment typically includes eradication of *H. pylori*, acid suppression, nutritional support, and lifestyle modification. This article highlights the importance of early recognition, individualized treatment strategies, and long-term surveillance in preventing complications and improving patient outcomes.

Keywords: Chronic gastritis; *Helicobacter pylori*; gastric mucosa; autoimmune gastritis; atrophic changes; proton pump inhibitors; gastric biopsy; gastrointestinal inflammation; vitamin B12 deficiency; gastric cancer prevention.

Chronic gastritis is a long-lasting inflammatory condition of the gastric mucosa that develops gradually and may persist for years, often without producing specific or alarming symptoms. Many patients do not seek medical attention until the condition has progressed significantly or until complications arise. The clinical course of chronic gastritis is shaped by its underlying cause, the patient's general health, environmental exposures, and comorbidities such as Helicobacter pylori infection or prolonged use of non-steroidal anti-inflammatory drugs (NSAIDs).

The early phase of chronic gastritis is often subtle. Patients may complain of vague discomfort in the upper abdomen, early satiety after meals, bloating, mild nausea, or intermittent heartburn. These symptoms can easily be mistaken for common digestive disturbances, leading to self-medication or delayed consultation. Over time, inflammation of the gastric lining can evolve into atrophic changes, where the glands that produce acid and enzymes begin to deteriorate. This process may result in hypochlorhydria (reduced stomach acid) and impaired digestion.

In cases of autoimmune gastritis, where the body produces antibodies that attack parietal cells or intrinsic factor, vitamin B12 deficiency becomes a serious concern. These patients may present not only with digestive complaints but also with neurological symptoms such as numbness, fatigue, or difficulty concentrating, all indicative of pernicious anemia. In contrast, gastritis associated with H. pylori tends to start in the antrum of the stomach and may progressively spread to other regions, causing widespread mucosal damage and increasing the risk of gastric ulcers or, in chronic cases, intestinal metaplasia and even gastric carcinoma. Diagnosis is often made through upper gastrointestinal endoscopy, during which the physician visually inspects the stomach lining and takes biopsies for histological analysis. The Sydney classification system is commonly used to evaluate the type and severity of gastritis, differentiating between superficial, atrophic, and metaplastic changes. Testing for H. pylori is

essential and can be done through urea breath tests, stool antigen assays, or biopsy-based methods.

The cornerstone of treatment lies in addressing the underlying cause. For patients with H. pylori -positive gastritis, eradication therapy is vital. This typically involves a combination of a proton pump inhibitor (to suppress acid) and two antibiotics, such as amoxicillin and clarithromycin, taken over 10 to 14 days. Eradicating the infection not only reduces inflammation but also prevents long-term complications. However, growing antibiotic resistance is a challenge, and therapy must be tailored accordingly, sometimes requiring quadruple therapy with bismuth-based agents.In cases unrelated to H. pylori, such as NSAID-induced or autoimmune gastritis, treatment strategies focus on reducing gastric acid, protecting the mucosal barrier, and managing associated deficiencies. Proton pump inhibitors are highly effective in healing erosions and reducing symptoms. For patients with anemia, iron or vitamin B12 supplementation may be necessary. Those with autoimmune gastritis often require lifelong parenteral vitamin B12, as intrinsic factor deficiency impairs oral absorption.

Lifestyle modifications form an essential component of therapy. Patients are encouraged to avoid alcohol, caffeine, spicy and acidic foods, smoking, and irregular eating habits. Managing stress, ensuring balanced nutrition, and maintaining regular mealtimes can greatly alleviate symptoms and support mucosal healing. In the long term, surveillance is important, especially for patients with atrophic gastritis or intestinal metaplasia, as they are at increased risk for gastric neoplasia. Periodic endoscopy may be recommended to monitor mucosal changes and intervene early if dysplasia is detected.

In conclusion, chronic gastritis is a multifaceted condition that requires a nuanced and patient-centered approach. Its clinical course is often slow but progressive, and symptoms may not always reflect the underlying severity. Effective management hinges on accurate diagnosis, elimination of contributing factors, pharmacological therapy, and consistent follow-up. While the condition is generally manageable, failure to recognize and treat it appropriately can lead to serious, even life-threatening, complications. Therefore, clinicians must maintain a high index of suspicion in patients with vague gastrointestinal complaints and implement evidence-based strategies to ensure optimal outcomes.

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