

A COMPREHENSIVE REVIEW OF GASTRITIS CLINICAL MANIFESTATION AND TREATMENT

ALAA ESSA ALMULLA
ABDULAZIZ HAMAD ALBAHRI
MANAL AHMED ALSUWAILIM
AMEERAH IBRAHEEM ALMREDEEF
AQEELH ABDULAZIZ SAID AL IBRAHIM
PRIMARY HEALTH CARE, NATIONAL GUARD-DAMMAM, DAMMAM, SAUDI ARABIA

MAHER ABDRAHALRASOOL ABDULAL
ABDULELAH HABIB BUKHAMSEEN
SUAD ABUDALLAH AL-NASSER
IMAM ABDULRAHMAN BIN FAISAL HOSPITAL, NATIONAL GUARD-DAMMAM, SAUDI ARABIA

Abstract:

Background:

Aim: This review aims to provide an in-depth exploration of the clinical manifestations and emerging therapeutic approaches for gastritis, drawing from recent research and clinical insights. **Method:** The PubMed and Google Scholar Search Engines were the primary databases used for the search process, with articles collected from 1980 to 2024. **Conclusion:** Acute and chronic gastritis present similar symptoms such as abdominal pain and nausea, yet their severity and duration differ. Acute gastritis typically leads to intense, temporary symptoms, whereas chronic gastritis causes persistent discomfort and a sense of fullness. Distinguishing between the two is crucial for accurate diagnosis and treatment, which depend on factors like mucosal alterations and blood gastrin levels. While acute gastritis can often be effectively managed with PPIs and supportive care, chronic gastritis necessitates a comprehensive strategy that incorporates antibiotics and ongoing management to reduce the likelihood of serious complications. The success of these treatment approaches has a direct impact on patient outcomes, underscoring the importance of accurate diagnosis and personalized therapeutic strategies for both types of gastritis.

Keywords: Gastritis, Clinical Manifestations, Therapeutic strategies and Diagnostic approaches

INTRODUCTION

Gastritis encompasses a range of inflammatory conditions affecting the gastric mucosa, which can be classified into acute and chronic forms, including erosive, non-erosive, and autoimmune gastritis. Acute gastritis is characterized by a sudden inflammation of the stomach lining, often triggered by irritants such as non-steroidal anti-inflammatory drugs (NSAIDs), alcohol, or stress. In contrast, chronic gastritis develops gradually and can persist for months or years, often resulting from long-term exposure to irritants or infections, particularly *Helicobacter pylori* (*H. pylori*). *H. pylori* infection is a significant risk factor for chronic gastritis, peptic ulcers, and gastric cancer, highlighting the importance of early detection and treatment. [1,2] Chronic gastritis can be further categorized into erosive and non-erosive types. Erosive gastritis involves the presence of gastric erosions, which can be caused by NSAIDs or other irritants, leading to symptoms such as abdominal pain and potential bleeding. Non-erosive gastritis, on the other hand, refers to inflammation without erosion, often associated with autoimmune conditions or infections. [3,4] Autoimmune gastritis is a specific form of chronic gastritis where the immune system mistakenly attacks the gastric mucosa, leading to the destruction of gastric parietal cells and resulting in conditions such as pernicious anemia. [5] This type of gastritis is characterized by the presence of autoantibodies against gastric H⁺/K⁺-ATPase and is often associated with vitamin B12 deficiency due to impaired intrinsic factor production. Atrophic gastritis, a subtype of chronic gastritis, involves the thinning of the gastric mucosa and is considered a precancerous condition for gastric cancer. [6] The prevalence of atrophic gastritis is notably higher in individuals infected with *H. pylori*, suggesting a strong correlation between this bacterium and the progression of gastric mucosal atrophy. [7]

CLINICAL MANIFESTATION OF GASTRITIS

Acute gastritis is characterized by inflammation of the stomach lining, resulting in a range of signs and symptoms that can significantly impact a patient's quality of life. The most common symptoms include gnawing or burning pain in the upper abdomen, which may fluctuate with eating. Nausea is another prevalent symptom, often accompanied by an inclination to vomit, which can sometimes include blood. Patients with acute gastritis frequently report a loss of appetite, which can be attributed to the discomfort associated with eating. Additionally, many experience a feeling of fullness after consuming only a small amount of food, further contributing to their reluctance to eat. Bloating, or a sensation of abdominal distension, is also commonly reported among those suffering from this condition. [8-10]

Endoscopic evaluations frequently reveal significant findings associated with acute gastritis. One common manifestation is acute erosive and hemorrhagic gastroduodenitis, which indicates inflammation and bleeding in the stomach and duodenum. This condition is often accompanied by highly active gastric inflammation, which can be identified in patients through the presence of gastric ulcers and erosions. Histopathological examinations of gastric biopsies from patients with acute gastritis often reveal intense neutrophil infiltrates, which are indicative of acute inflammation. The presence of microabscesses within the gastric tissue further supports the diagnosis, as these localized areas of infection and inflammation are characteristic of acute gastritis. Additionally, multiple erosions on the gastric lining can be observed, which are specific manifestations of the condition. The severity of acute gastritis can be classified histologically, with grades two and three indicating significant inflammation. This classification is crucial for understanding the extent of the disease and guiding treatment options. In cases where large spiral-shaped bacteria are identified, they may serve as a causative infectious agent of acute neutrophilic gastritis, highlighting the role of infectious agents in the pathogenesis of the condition. [7,10,11]

Chronic gastritis is characterized by a range of signs and symptoms that can significantly impact a patient's quality of life. One of the most common symptoms is burning abdominal pain, typically located in the upper middle abdomen, which often worsens after meals. This discomfort can be accompanied by persistent upper abdominal discomfort, a key indicator of the condition. Patients frequently report nausea and vomiting, which may sometimes include blood, indicating severe issues with the stomach lining. These symptoms can be distressing and may lead to further complications if not addressed. Additionally, individuals with chronic gastritis often experience bloating and a feeling of fullness after consuming small amounts of food, which can contribute to a decreased appetite. This loss of appetite can lead to unintentional weight loss, further compounding the health challenges faced by these patients. Indigestion, characterized by a gnawing or burning sensation in the upper abdomen, is another common symptom associated with chronic gastritis. Patients may also experience vague dyspeptic symptoms, which encompass a range of discomforts in the upper abdomen that are exacerbated by dietary factors, making them a direct answer to inquiries about the signs and symptoms of this condition. Acid regurgitation is also prevalent in these patients, as the backflow of stomach acid into the esophagus can lead to significant discomfort. Furthermore, early satiety, the feeling of being full after consuming only a small amount of food, is often reported. In more severe cases, the presence of blood in vomit or stool can indicate serious complications such as ulcers or erosive gastritis, necessitating immediate medical attention. [9,12] The combination of these symptoms can lead to significant distress and may require supportive treatment to manage the condition effectively.

Both forms of gastritis share common symptoms, such as stomach pain, nausea, and indigestion, but the severity and duration can differ significantly. Acute gastritis typically presents with more intense, short-lived symptoms, while chronic gastritis may lead to a dull, persistent discomfort and a feeling of fullness after eating. [9] Differentiating between acute and chronic gastritis is crucial for effective diagnosis and treatment. Several key factors can aid in this differentiation, including mucosal changes, hydrochloric acid secretion levels, blood gastrin levels, and specific histological features. Firstly, the classification of mucosal changes observed during endoscopy is a significant indicator. Mucosal alterations can be categorized into slight, moderate, and advanced changes. Acute gastritis typically presents with slight changes, while chronic gastritis is associated with moderate to advanced alterations, which correlate with decreased hydrochloric acid secretion. In chronic gastritis, particularly with advanced atrophic changes, patients often exhibit significantly lower peak acidity and acid output compared to those with acute gastritis. Blood gastrin levels also play a critical role in distinguishing between the two forms of gastritis. Patients with chronic gastritis, especially those over 60 years with advanced atrophic changes, tend to have abnormally high blood gastrin levels. This contrasts with acute gastritis, where such elevations are less common. The relationship between hydrochloric acid secretion and blood gastrin levels further supports this differentiation; cases of achlorhydria or hypochlorhydria are more frequently observed in chronic gastritis, indicating a functional impairment of gastric acid secretion. [12,13]

Histological examination through gastric mucosal biopsy is another essential method for differentiation. Chronic gastritis is characterized by specific histological features such as intestinal metaplasia and atrophic changes, which are less prevalent in acute gastritis. The presence of intestinal metaplasia, in particular, is a hallmark of chronic gastritis and can be identified through enzyme-histochemical studies. [12,13] Additionally, serological tests for antibodies can provide further insights. The presence of parietal cell antibodies may

indicate autoimmune gastritis, a form of chronic gastritis, while intrinsic factor antibodies can suggest chronic gastritis related to pernicious anemia. Testing for *Helicobacter pylori* antibodies is also critical, as this bacterium is more commonly associated with chronic gastritis and can lead to complications such as gastric cancer [14]

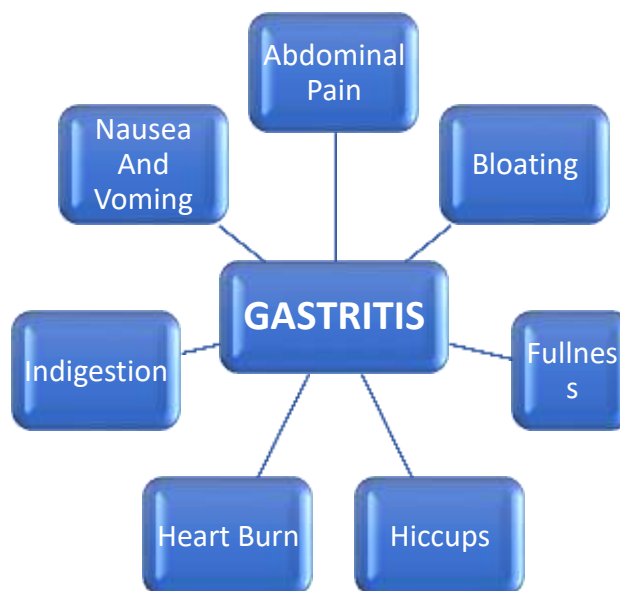


FIGURE (1): CLINICAL MANIFESTATION OF GASTRITIS.

TREATMENT

The treatment approaches for acute and chronic gastritis differ significantly and vary due to their distinct underlying causes and clinical presentations. Acute gastritis treatment primarily focuses on alleviating symptoms and addressing the underlying cause. For instance, if acute gastritis is associated with an infection by *Helicobacter pylori*, eradication therapy is recommended, which typically includes a combination of proton pump inhibitors (PPIs). PPIs, such as lansoprazole, are effective in reducing stomach acid and promoting healing of the gastric lining. In cases where NSAIDs are the culprit, discontinuation of the offending medication is crucial. Supportive care, including dietary modifications to avoid irritants and stress, is also advised to manage symptoms. [4,9,15] In many cases, acute gastritis resolves quickly with appropriate treatment, leading to favorable patient outcomes, including symptom relief and prevention of complications.

In contrast, chronic gastritis is a long-term condition often linked to persistent *H. pylori* infection or autoimmune disorders. The treatment for chronic gastritis usually involves a combination of antibiotics to eradicate *H. pylori* and PPIs to manage acid production. This dual approach is crucial, as untreated chronic gastritis can lead to severe complications, including peptic ulcers, gastrointestinal bleeding, and an increased risk of gastric cancer. The management of chronic gastritis is more complex and supportive in nature, requiring ongoing monitoring and treatment adjustments that can impact patient adherence and overall outcomes. While eradication of *H. pylori* is also a goal in chronic gastritis, the treatment may involve long-term management strategies, including dietary changes and monitoring for potential complications. [12,16] Autoimmune gastritis, a specific type of chronic gastritis, may require additional interventions to address vitamin deficiencies, particularly vitamin B12, due to the loss of intrinsic factor production. [17]

The differences in treatment approaches also reflect the underlying pathophysiology of each condition. Acute gastritis is often self-limiting, and timely intervention can lead to complete recovery. In contrast, chronic gastritis necessitates a more comprehensive strategy to address the persistent nature of the disease and its complications. The presence of *H. pylori* infection in chronic cases underscores the importance of targeted antibiotic therapy, as eradication of the bacteria is essential for preventing further gastric damage and improving long-term outcomes.

CONCLUSION

Acute and chronic gastritis present similar symptoms such as abdominal pain and nausea, yet their severity and duration differ. Acute gastritis typically leads to intense, temporary symptoms, whereas chronic gastritis causes persistent discomfort and a sense of fullness. Distinguishing between the two is crucial for accurate diagnosis and treatment, which depend on factors like mucosal alterations and blood gastrin levels. While acute gastritis can often be effectively managed with PPIs and supportive care, chronic gastritis necessitates a comprehensive strategy that incorporates antibiotics and ongoing management to reduce the likelihood of serious

complications. The success of these treatment approaches has a direct impact on patient outcomes, underscoring the importance of accurate diagnosis and personalized therapeutic strategies for both types of gastritis.

ACKNOWLEDGEMENT

The authors would like to express their gratitude to the publicly accessible online library databases for their invaluable assistance in finishing an extensive literature study. Lastly, the authors would like to sincerely thank the corresponding author for their informative remarks, which greatly improved the quality of the paper.

AUTHOR CONTRIBUTIONS

Although all authors made substantial contributions through data collection and literature searches, the original author created the text. Each author took full responsibility for the work, participated in the manuscript's critical review, and approved the final draft.

CONFLICT OF INTEREST

Authors declare they don't have any conflict of interest.

ETHICAL APPROVAL

Not Applicable

REFERENCES

1. Cheli R, Perasso A, Giacosa A, Cheli R, Perasso A, Giacosa A: Definition of Gastritis. *Gastritis: A Critical Review*. 1987:3-11.
2. Araya J, Villaseca M, Roa I, Roa J: Helicobacter pylori and chronic gastritis: relationship between infection and inflammatory activity in a high risk population for gastric cancer. *Revista Medica de Chile*. 2000, 128:259-265.
3. Gallagher CG, Lennon JR, Crowe JP: Chronic erosive gastritis: a clinical study. *American Journal of Gastroenterology (Springer Nature)*. 1987, 82.
4. Hunt R: The role of Helicobacter pylori in pathogenesis: the spectrum of clinical outcomes. *Scandinavian Journal of Gastroenterology*. 1996, 31:3-9.
5. Sekino T, Hino T, Saito Y, Abe S, Shibuya T: Studies on the auto-immune antibody in patients with gastritis. *Gastroenterologia Japonica*. 1968, 3:192-192.
6. Carboni M, Guadagni S, Pistoia M, et al.: Chronic atrophic gastritis and risk of N-nitroso compounds carcinogenesis. *Langenbeck's Archives of Surgery*. 1988, 373:82-90.
7. Yang H, Dixon MF, Li X, Xu Z, Zhou D, Blum AL: Acute Gastritis Associated with Infection of Large Spiral-Shaped Bacteria. *American Journal of Gastroenterology (Springer Nature)*. 1995, 90.
8. Collins S: Gastritis and Altered Motility; the Ability of a Mucosal Inflammatory Reaction to Alter Enteric Nerve and Smooth Muscle in the Gut. *Helicobacter pylori, gastritis and peptic ulcer*. Springer; 1990. 370-374.
9. Marcial G, Rodríguez C, Medici M, de Valdez GF: New approaches in gastritis treatment. *Gastritis and gastric cancer-new insights in gastroprotection, diagnosis and treatments*. 2011:153-176.
10. Boehme MW, Autschbach F, Ell C, Raeth U: Prevalence of silent gastric ulcer, erosions or severe acute gastritis in patients with type 2 diabetes mellitus--a cross-sectional study. *Hepato-gastroenterology*. 2007, 54:643-648.
11. Caletti G, Fusaroli P, Tucci A, Fedrigo M, Bettini G, Roda E: Severe acute gastritis associated with Helicobacter pylori infection. *Digestive and Liver Disease*. 2000, 32:34-38.
12. Chatterjee D: Idiopathic chronic gastritis. *Surgery, Gynecology & Obstetrics*. 1976, 143:986-1000.
13. Wada T, Sato K, Kinoshita H, Fujita I, Sugawara M, Ikeshita T: Studies on chronic gastritis A comparative study of histology and gastric juice analysis. *Gastroenterologia Japonica*. 1968, 3:190-190.
14. Desai H: Investigations proposed to accurately classify chronic gastritis. *The Journal of the Association of Physicians of India*. 2007, 55:293-296.
15. Rahden B, Scheurlen M, Filser J, Stein H, Germer C-T: Neu erkannte Nebenwirkungen von Protonenpumpeninhibitoren. *Der Chirurg*. 2012, 83.
16. Compare D, Pica L, Rocco A, et al.: Effects of long-term PPI treatment on producing bowel symptoms and SIBO. *European journal of clinical investigation*. 2011, 41:380-386.
17. Toh B-H, Biondo M, Marshall A, Greenwood D, SENTRY J, Alderuccio F: The gastric H/K ATPase in the pathogenesis of autoimmune gastritis. *Mechanisms and Consequences of Proton Transport*. 2002:107-114.