GASTRITS: ETIOLOGY AND TREATMENT

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https://doi.org/10.5281/zenodo.15070415

Abstract. *Gastritis refers to the inflammation of the gastric mucosa and is often used to describe the abnormal appearance of abnormal gastric mucosa on endoscopy or radiology. Gastritis encompasses infectious or immunological inflammation of the gastric mucosa and the host's response. Histopathological evidence of inflammation in the stomach lining is essential to diagnose this condition. Gastropathy is characterized as a gastric mucosal disorder without inflammation, often featuring epithelial injury and subsequent regeneration. Gastritis and gastropathy are not mutually exclusive conditions and might sometimes coexist. In clinical practice, gastritis may be accompanied by signs of mucosal injury, whereas gastropathy may show some evidence of an inflammatory reaction in the gastric mucosa. Gastritis can be classified based on the acuity of the condition (acute versus chronic), the histological features of the inflammation, or its etiology. Although there is no universally accepted categorization and classification of gastritis, it is crucial to understand the histological characteristics and etiological factors associated with the different types of gastritis to comprehend their presentation and classification. Appropriate histological evaluation is also essential in devising management plans for this disease. This review discusses the histological and morphological presentations of gastritis, assesses their prognostic significance, and outlines the guideline-recommended management approaches for these conditions. The primary objective of this topic is to improve patient outcomes by enhancing the competence of healthcare providers.*

Keywords: *Inflammation, gastropathy, mucosal disorder, epithelial injury, subsequent regeneration, inflammatory reaction.*

ГАСТРИТЫ: ЭТИОЛОГИЯ И ЛЕЧЕНИЕ

Аннотация. *Гастрит относится к воспалению слизистой оболочки желудка и часто используется для описания аномального вида аномальной слизистой оболочки желудка при эндоскопии или рентгенологии. Гастрит охватывает инфекционное или иммунологическое воспаление слизистой оболочки желудка и реакцию хозяина. Гистопатологические доказательства воспаления в слизистой оболочке желудка необходимы для диагностики этого состояния. Гастропатия характеризуется как расстройство слизистой оболочки желудка без воспаления, часто с повреждением эпителия и последующей регенерацией. Гастрит и гастропатия не являются взаимоисключающими состояниями и иногда могут сосуществовать. В клинической*

практике гастрит может сопровождаться признаками повреждения слизистой оболочки, тогда как гастропатия может показывать некоторые признаки воспалительной реакции в слизистой оболочке желудка. Гастрит можно классифицировать на основе остроты состояния (острый или хронический), гистологических особенностей воспаления или его этиологии. Хотя не существует общепринятой категоризации и классификации гастрита, крайне важно понимать гистологические характеристики и этиологические факторы, связанные с различными типами гастрита, чтобы понять их проявления и классификацию. Соответствующая гистологическая оценка также имеет важное значение при разработке планов лечения этого заболевания. В этом обзоре обсуждаются гистологические и морфологические проявления гастрита, оценивается их прогностическое значение и излагаются рекомендуемые в руководствах подходы к лечению этих состояний. Основной целью этой темы является улучшение результатов лечения пациентов путем повышения компетентности поставщиков медицинских услуг.

Ключевые слова: *воспаление, гастропатия, нарушение слизистой оболочки, повреждение эпителия, последующая регенерация, воспалительная реакция.*

Introduction

Gastritis is the inflammation of the gastric mucosa and is often used to describe the abnormal appearance of abnormal gastric mucosa on endoscopy or radiology. Gastritis encompasses infectious or immunological inflammation of the gastric mucosa and the host response. Histopathological evidence of inflammation in the stomach lining is essential to diagnose this condition. Gastropathy is a gastric mucosal disorder without inflammation, featuring epithelial injury and subsequent regeneration. Gastritis and gastropathy are not mutually exclusive conditions and might sometimes coexist. In clinical practice, gastritis may be accompanied by signs of mucosal injury, whereas gastropathy may present with an inflammatory reaction in the gastric mucosa.

Gastritis is classified based on the acuity of the condition (acute versus chronic), the histological features of inflammation, or the etiology. Although the categorization and classification of gastritis are not universally accepted, understanding the histological characteristics and etiological factors associated with the different types of gastritis is essential.

Appropriate histological evaluation is also essential in devising management plans for this disease. The primary objective is to equip treating clinicians with the ability to improve patient outcomes through early intervention.

Etiology

Acute Gastritis

Acute gastritis is temporary stomach lining inflammation caused by stress on the gastric mucosa, manifesting as either hemorrhagic or non-hemorrhagic symptoms. This condition can develop due to various factors, including uremia, ischemia, shock, corrosive agents, medications, radiation, trauma, severe burns, sepsis, or alkaline-bile reflux. Certain infections, such as enteroviruses, can also cause a self-limited episode of gastritis. Acute gastritis may result from reduced gastric mucus secretion, mucosal barrier disruption, or decreased mucosal blood flow, depending on the underlying cause.

Chronic Gastritis

Chronic gastritis is categorized into 2 forms—atrophic and non-atrophic. The primary cause of chronic gastritis is a *Helicobacter pylori* infection, which typically starts with a non-atrophic morphology. The non-atrophic form of chronic gastritis can progress to atrophic without treatment. The most common cause of atrophic chronic gastritis is autoimmune gastritis, though the etiology remains unclear. Autoimmune gastritis exhibits a chronic mononuclear inflammation accompanied by severe atrophic gastritis, which usually affects the corpus, along with the presence of autoantibodies against parietal cells or the intrinsic factor. However, whether autoimmune gastritis is an independent disorder or if an *H pylori* infection triggers the autoimmune response in susceptible individuals is unclear.

Reactive Gastritis

Reactive gastritis or gastropathy has numerous causative factors with acute gastritis.

Reactive gastritis may be caused by specific medications, alcohol consumption, radiation exposure, and duodenal (bile) reflux. These causative agents lead to histological mucosal lesions characterized by low-grade inflammation of the gastric mucosa. Although usually asymptomatic, they are revealed through endoscopy, often showing multiple erosions or ulcers without signs of atrophic changes. The use of immune checkpoint inhibitors to treat various malignancies has contributed to the incidence of reactive gastritis, although the condition remains considerably rare.

The Sydney System of Classification for Gastritis

The Histological Division of the Sydney System was introduced in 1990 and has since become the most widely cited classification system for the morphological features of gastritis in endoscopic biopsies. This system conveys information about the type, severity, and extent of gastric pathology. The classification system conveys the topography of gastritis, which can be restricted to the antrum or corpus or involve the entire stomach (pan gastritis).

If the etiology of the disease is known, this is added as a prefix to denote the topography.

For instance, the label "autoimmune corpus gastritis" is used if the disease is autoimmune. The Sydney System of Classification further delineates 5 graded morphological variables that may be added as a suffix to the core topography. These variables include the type or chronicity of inflammation, gastritis activity, intestinal metaplasia, the extent of atrophy, and the presence or absence of *Heliobacter pylori*. The morphological features are graded as absent, mild, moderate, or severe. The Sydney System of Classification recommends at least 2 random biopsies from both the antrum and corpus, along with an additional biopsy from the incisura angularis. Although the classification system provides a standardized and concise means of documenting the extent and severity of gastritis, the method for predicting or forecasting future morphological changes is impossible.

Classification of Gastritis Based on Etiological Factors

An alternative approach to classifying gastritis considers the etiology and chronicity of the inflammation. This approach categorizes gastritis into 3 main subtypes—acute, chronic, and special. Infectious gastritis is most commonly attributed to the global prevalence of *H pylori* infection. Other types of infectious gastritis include phlegmonous gastritis (caused by pyogenic bacteria), mycobacterial gastritis (caused by *Mycobacterium tuberculosis*), syphilitic gastritis, viral gastritis (caused by cytomegalovirus and herpes simplex virus).

Granulomatous gastritis is a special gastritis observed in patients with Crohn disease and sarcoidosis. Lymphocytic gastritis, collagenous gastritis, and eosinophilic gastritis are additional special subtypes of gastritis with unclear etiologies. Lymphocytic and collagenous gastritis have been associated with celiac disease, whereas eosinophilic gastritis has a strong connection to atopic conditions and food allergens.

According to the 2015 Kyoto Consensus Conference, a classification of gastritis based on etiological factors is outlined as follows:

- Autoimmune gastritis
- Infectious gastritis
  - Gastric phlegmon
  - Bacterial gastritis
    - *H pylori*-induced
    - Enterococcal
    - Mycobacterial
  - Viral gastritis
    - Cytomegaloviral
    - Enteroviral
  - Fungal gastritis

- Parasitic gastritis
  - Gastric anisakiasis
  - Cryptosporidium
  - Gastric *Strongyloides stercoralis*
- Gastritis due to other diseases
  - Crohn disease
  - Sarcoidosis
  - Vasculitis
- Gastritis due to external causes
  - Alcoholism
  - Radiation
  - Chemicals
- Special gastritis
  - Allergic gastritis
  - Gastritis due to biliary reflux
  - Lymphocytic gastritis
  - Ménétrier disease
  - Eosinophilic gastritis

Epidemiology

Determining the incidence of acute gastritis can be challenging due to the common causes, such as enterovirus infections, which typically result in mild and self-limited episodes that go unreported. Other factors leading to acute gastritis, such as sepsis, ischemia, and caustic injury, are relatively rare compared to chronic *H pylori*–associated gastritis and chronic atrophic (autoimmune) gastritis. Recent data demonstrates chronic atrophic gastritis is estimated to affect approximately 25% of the global population. Furthermore, the risk of developing chronic atrophic gastritis is about 2.4 times higher in patients with *H pylori.*

In Western populations, a declining incidence of infectious gastritis is thought to be caused by an increasing prevalence of autoimmune gastritis. Autoimmune gastritis is more prevalent in women and older individuals, with estimated rates ranging from 2,5% to 5,5%.

However, the available data may have limited reliability. Chronic *H pylori*–associated nonatrophic gastritis continues to be highly prevalent in developing countries. In Western populations, the prevalence of *H pylori* infection in children is approximately 15%, whereas the prevalence is 54% in developing countries. The prevalence of *H pylori* infection in developing countries varies significantly based on geographical region and socioeconomic conditions.

Treatment / Management

Approach to Treatment

As mentioned earlier, eradicating *H pylori* is recommended for all patients with evidence of gastritis on diagnostic testing. Furthermore, eradication therapy is the initial treatment option for patients with dyspepsia who have a documented *H pylori* infection. In addition, *H pylori* eradication therapy is indicated in patients with peptic ulcer disease, functional dyspepsia, idiopathic thrombocytopenic purpura (ITP), unexplained iron-deficiency anemia, and in cases where long-term nonsteroidal anti-inflammatory drug treatment is anticipated, particularly in patients with a history of peptic ulcer disease. In patients with functional dyspepsia, eradication therapy has a limited impact on symptom relief. Nonetheless, the therapy is advantageous in mitigating the risk of peptic ulcer disease.

Eradication therapy for *H pylori* in patients with non-atrophic chronic gastritis is highly recommended to promote healing and reduce the risk of gastric cancer. For patients with atrophic gastritis, eradication therapy targeted at the organism may result in partial regression of the gastritis and offer some potential benefits. Although the eradication therapy in patients with intestinal metaplasia does not reverse the metaplastic changes, the progression to neoplasia is slowed without reducing the overall risk of gastric cancer. Therefore, a cautious or weak recommendation is considered in this context.

The management of chronic gastritis in patients who initially test negative for *H pylori* lacks standardized guidelines and tends to exhibit significant variability. Empirical use of proton-pump inhibitors (PPIs) has demonstrated effectiveness in alleviating symptoms for these patients. According to current guidelines, empiric PPI therapy is recommended for individuals aged younger than 60 with dyspepsia if they test negative for *H pylori* or experience persistent symptoms despite undergoing eradication therapy. Patients who do not experience relief from these treatments may be considered for prokinetic therapy or tricyclic antidepressants. Notably, the supporting evidence is low-to-moderate quality.

Currently, definitive treatment does not exist for patients with atrophic gastritis. The pivotal aspect in treating patients with atrophic gastritis is the application of risk stratification systems to assess the severity of the disease and determine the risk of gastric malignancies. For this purpose, utilizing the Operative Link on Gastritis Assessment (OLGA) and Operative Link on Gastric Intestinal Metaplasia Assessment (OLGIM) grading systems is recommended. Staging gastritis using the OLGA and OLGIM systems, with a stage III or IV classification, is associated with a significantly elevated risk of gastric cancer. This approach provides an easily translated method for assessing attributable risk.

These systems incorporate atrophy scores obtained through histological assessment of gastric biopsies and consider atrophy topography to assign clinical stages.

Histological Grading of Gastritis

In normal gastric mucosa, an acceptable range is typically 3 to 6 lymphocytes, plasma cells, or macrophages per high power field, or 2 to 3 cells located between foveolae. The degree of increase from these numbers determines the severity of gastritis, which is graded as mild (+--), moderate (++-), or marked (+++). Notably, this density measurement should be performed away from any lymphoid follicles, as they could be related to an underlying *H pylori* infection. Lymphocytic gastritis occurs when more than 25 lymphocytes are observed per 100 epithelial cells within the glandular epithelium. The density of neutrophils measures the activity of gastritis. The grading of gastritis activity is followed as neutrophils in the lamina propria indicate mild (+--) activity, neutrophils within the epithelium denote moderate (++-) activity, and neutrophils in the glandular lumen signify marked (+++) activity.

The discrepancy between the expected glands for the anatomical site of the gastric mucosa and what is observed represents atrophy. A reduction or complete absence of glandular units leads to collagen deposition in the lamina propria. Metaplastic changes involve the replacement of normal glandular units with metaplastic and/or dysplastic units. A score of 1 is allocated when a 2% to 35% loss of the glandular architecture or its metaplastic transformation occurs, a score of 2 is assigned for a 35% to 67% loss, and a score of 3 is designated for a loss exceeding 65%. The OLGA staging system categorizes gastritis into 5 stages, each associated with a progressively higher risk of cancer, determined by the atrophy score. In addition, an overall atrophy score based on topography is assigned, and these scores are tallied to determine the corresponding OLGA stage. Although the OLGIM staging system relies solely on intestinal metaplasia for the atrophy score, enhancing inter-observer reproducibility, a notable decrease in sensitivity for identifying high-risk patients is apparent.

Patients classified as OLGA/OLGIM stage III or IV face a considerable risk of developing gastric adenocarcinoma. As a result, regular surveillance endoscopy is strongly recommended for these individuals to enhance the chances of detecting gastric cancer in the early stages, enabling surgical treatment. The AGA recommends endoscopic surveillance every 3 years in these patients. Other clinical factors that should be considered when determining the frequency of surveillance include a family history of gastric cancer, residence in regions with a high incidence of gastric cancer, a history of persistent *H pylori* infection, smoking history, and dietary factors.

Conclusion

According to the Kyoto Global Consensus Conference, etiology is taken for reference in the classification of gastritis. The etiological picture of long-standing gastritis can include both environmental (e.g. *H. pylori*) and host-related (e.g. Autoimmunity) agents, potentially resulting in the atrophic transformation of native gastric mucosa. Epidemiological evidence implicates the atrophic microenvironment in *H. pylori* gastritis as a major factor responsible for the etiopathogenesis of more than 85% of gastric malignancies.

The atrophic transformation of gastric mucosa gives rise to different histological phenotypes, all of which have been biologically profiled and can be histologically scored. They may also be associated with a range of functional changes, which can serve as (quantitative) serological markers of the atrophic process. It is easy to imagine the atrophy-remodeled gastric microbiota having a role as co-promoter in the atrophic cancer-prone microenvironment.

Over the coming years, we will see how this multidisciplinary approach can be optimized for the purpose of designing global strategies for eradicating gastric cancer and implementing patient-tailored prevention strategies. The available evidence does suggest that combining primary and secondary prevention strategies can realistically succeed in cutting the epidemiological impact of gastric cancer – the world's fourth leading cause of cancer-related death.

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