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REVIEW ARTICLE

Erosive gastritis and duodenitis: causes, diagnosis and treatment

Genaro Vázquez-Elizondo

Department of Gastroenterology, Centro de Enfermedades Digestivas NCARE/GastroAlliance, Monterrey, Nuevo Leon, Mexico

Abstract

Gastritis is a condition encompassing a spectrum of gastric mucosal lesions, and typically is characterized by changes and usually erosions. It frequently causes symptoms such as epigastric pain, dyspeptic syndromes, and occasionally upper gastrointestinal bleeding. The term comes from the Greek words *gastḗr gastrós*, meaning "inflammatory process (inflammation/heat) affecting the stomach." Etiology includes infectious causes (e.g., *Helicobacter pylori*), drug-induced lesions (e.g., nonsteroidal anti-inflammatory drugs), alcohol, autoimmune diseases (autoimmune gastritis), and other less frequent causes. Diagnosis usually involves targeted testing and upper endoscopy with biopsies to stratify and identify the etiology. Treatment is typically directed at the causative agent, as appropriate, in conjunction with acid suppression using proton pump inhibitors or, in individualized contexts, selective potassium-competitive acid blockers. Since clinically and etiopathogenically gastritis shares mechanisms and is indistinguishable from duodenal inflammation (duodenitis), this revision considers both entities from a pragmatic point of view and narratively synthesizes a practical, evidence-based approach to addressing this issue.

Keywords: Gastritis. Duodenitis. Treatment. Diagnosis. Drugs. Pathology.

Gastritis y duodenitis erosivas: causas, diagnóstico y tratamiento

Resumen

La gastritis corresponde a un espectro de lesiones de la mucosa gastrica y se caracteriza tipicamente por cambios y usualmente erosiones. Con frecuencia condiciona la presencia de sintomas como dolor epigastrico, sindromes dispepticos y en ocasiones sangrado digestivo alto. El termino proviene del griego *gastḗr gastrós*, que significa ≪proceso flogistico (inflamacion/calor) afectando al estomago≫. La etiologia incluye causas infecciosas (p. ej., *Helicobacter pylori*), lesiones por farmacos (p. ej., antiinflamatorios no esteroideos), alcohol y enfermedades autoinmunitarias (gastritis autoinmunitaria), entre otras menos frecuentes. El diagnostico suele incluir la realizacion de una evaluacion con pruebas dirigidas, asi como endoscopia superior con toma de biopsias con el fin de estratificar e identificar la etiologia. El tratamiento se dirige al agente causal segun corresponda en conjunto con el uso de supresion acida con inhibidores de la bomba de protones o, en contextos individualizados, bloqueadores selectivos del acido competitivos con potasio. Dado que desde el punto de vista clinico y etiopatogenico la gastritis comparte mecanismos y es indistinguible de la inflamacion duodenal (duodenitis), en esta revision se consideran ambas afecciones desde un punto de vista pragmatico y se sintetiza narrativamente un enfoque practico basado en la evidencia para su abordaje.

Palabras clave: Gastritis. Duodenitis. Tratamiento. Diagnóstico. Fármacos. Patología.

Correspondence:
Genaro Vázquez-Elizondo
E-mail: drgenarovazquez@gmail.com

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Introduction

Gastritis is defined by the histological presence of inflammation in the gastric mucosa produced by infectious agents or autoimmune causes, as well as the patient's response to these lesions. It should be mentioned that epithelial injury in the mucosa (with or without regeneration) without inflammation (host response) is termed "gastropathy"; however, these two phenomena may coexist and clinically usually present similarly. The first description of gastritis emerged in the 19th century as a result of autopsy analysis, and in 1947 it was categorized as acute and chronic.[1] Although there were different stratification systems, such as the Schindler classification, with the advent of evidence of *Helicobacter pylori* as a causative agent of gastritis in 1983, progress was made toward an integrated classification of endoscopic, histological, and etiological findings: the Sydney classification.[2]

Gastritis can be classified according to temporality, histological characteristics, and etiology, but there is no universally accepted classification, and for the purposes of clinical practice, it is essential to understand the pathophysiological mechanisms in order to provide a clinical management plan. This review will address gastritis from an etiological, clinical, and general therapeutic point of view.

Methodology

A narrative review focused on evidence published in international guidelines and consensus statements from the last 5 years was conducted, focusing on definition, etiology, and pharmacological treatment strategies.

Etiology and classification of histopathological findings

The etiological approach to gastritis classifies it into three main groups: acute, chronic, and special. However, with the integration of clinical context, endoscopic findings, and etiology, the classifications shown in tables 1 and 2 can be considered. Furthermore, histological classification is sometimes challenging for the clinician to understand in decision-making. Table 3 shows the classification derived from the revision of the Sydney system in 1994[3,4] according to the main findings obtained in histopathology reports, which may vary according to the etiology and temporality of gastritis. Of particular interest is the presence of atrophic changes with disappearance or substitution of gland types toward gastric dysplasia; thus, the report in these cases should employ the OLGA (Operative Link Gastritis Assessment) system,[5,6] shown in table 4; stages III and IV are particularly important, as they carry considerable risk for the development of gastric cancer and require the implementation of an endoscopic surveillance program.[7]

Table 1. Etiology of gastritis

Induced by *Helicobacter pylori*	Special types of gastritis Allergic Secondary to bile reflux Lymphocytic Eosinophilic Ménétrier disease
Induced by medications Nonsteroidal anti-inflammatory drugs Iron supplements Bisphosphonates	
Autoimmune	Secondary to external causes Alcohol Radiation Chemical
Stress-mediated Sepsis Hypovolemia Drug abuse (cocaine)	
Infectious Pyogenic (phlegmon)	Crohn disease
	Sarcoidosis
Bacterial Enterococci Mycobacteria (tuberculosis and non-tuberculosis) Syphilis	Vasculitis
Viral Cytomegalovirus Enterovirus	
Fungal Mucormycosis Candidiasis Histoplasmosis	
Parasitic Anisakiasis *Cryptosporidium* *Strongyloides stercoralis*	

Adapted from Jensen[1] and Sugano et al.[12].

Diagnosis

The clinical presentation of gastritis is usually conditioned by the clinical and etiological context in

Table 2. Classification of gastritis and examples of etiology

Group	Etiology
Acute	Drugs, stress-induced, uremia, ischemia, shock, corrosive agents, radiation, irritative (foods), sepsis, trauma, bacterial and viral infections, alcohol use, burns, bile/alkaline reflux, major surgery, organ failure, portal hypertension, congestive heart failure, increased intracranial pressure
Reactive gastropathy (chemical)	Endotoxic (bile/alkaline reflux, uremia) Exotoxic (nonsteroidal anti-inflammatory drugs, alcohol) Stress-induced
Chronic	*Helicobacter pylori* (and *Helicobacter heilmannii*) Autoimmune Chronic *H. pylori*-negative gastritis
Special	Lymphocytic Collagenous Eosinophilic Radiation Graft-versus-host disease Bacterial (syphilis, tuberculosis) Viral (cytomegalovirus, herpes simplex virus) Fungal (*Candida*, *Aspergillus*, *Mucormycosis*, *Coccidioides*, *Histoplasma*, *Cryptococcus*, *Pneumocystis*) Parasitic (*Anisakis*, *Cryptosporidium*, *Ascaris lumbricoides*, *Giardia*, *Toxoplasma*, *Schistosoma*)
Granulomatous	Idiopathic Crohn disease Sarcoidosis Barium granulomas
Hypertrophic	Ménétrier disease Zollinger-Ellison syndrome Hypertrophic Hypersecretory (protein-losing enteropathy)
Gastric vasculopathies	Ischemic Gastric antral vascular ectasia (watermelon stomach) Portal hypertensive gastropathy (congestive gastropathy) Varices Angiodysplasia Dieulafoy lesion Telangiectasias associated with hemodialysis
Gastric involvement in systemic diseases	Inflammatory bowel disease Amyloidosis Diabetes Mastocytosis Sjögren syndrome Hypercalcemia Siderosis

Translated from Pennelli et al.[12].

which a given patient is evaluated, although it is usually nonspecific and usually not determinative of etiology. Typical symptoms usually include epigastric pain or burning, fullness, early satiety, nausea, and vomiting, although occasionally it may be found incidentally during endoscopic evaluation. Thus, the differential diagnosis includes conditions such as functional dyspepsia, peptic ulcer disease, cholecystitis, pancreatitis, myocardial infarction, and celiac disease, among other upper gastrointestinal tract pathologies. In this way, diagnostic management and approach will be largely conditioned by the clinical context and, above all, by the presence of alarm signs or symptoms, some of which are age over 55 years, unexplained weight loss, presence of anemia, melena, persistent vomiting, and family history of gastric cancer.[8,9]

Upper endoscopy is a fundamental test in the diagnosis of gastritis, since without it, it is not possible to obtain the histopathological specimens that allow differential diagnosis of gastritis. Although it is essential to provide a description and photographic documentation of all segments and findings during the study (such as presence of focal lesions, fold thickening, polyps, vasculature, masses, or erosions) (Fig. 1), these findings usually have limited value because there is great interobserver variability.[10] Furthermore, it has been shown that the sensitivity of endoscopic findings in relation to the histopathological diagnosis of gastritis or *H. pylori* infection is approximately 57%.[11] On the other hand, although there may be local variability in the number of biopsies (according to available resources and technology, as well as clinical context), it is generally recommended to follow the standard Sydney protocol for performing biopsies, according to which one or two biopsies are taken from five gastric anatomical zones: greater curvature of the antrum, lesser curvature of the antrum, angular incisure, greater curvature of the body, and lesser curvature of the body (Fig. 2). This protocol usually allows optimization of etiological diagnosis.

Treatment

Regarding the treatment of erosive gastritis, we must recognize that, although the etiology may be

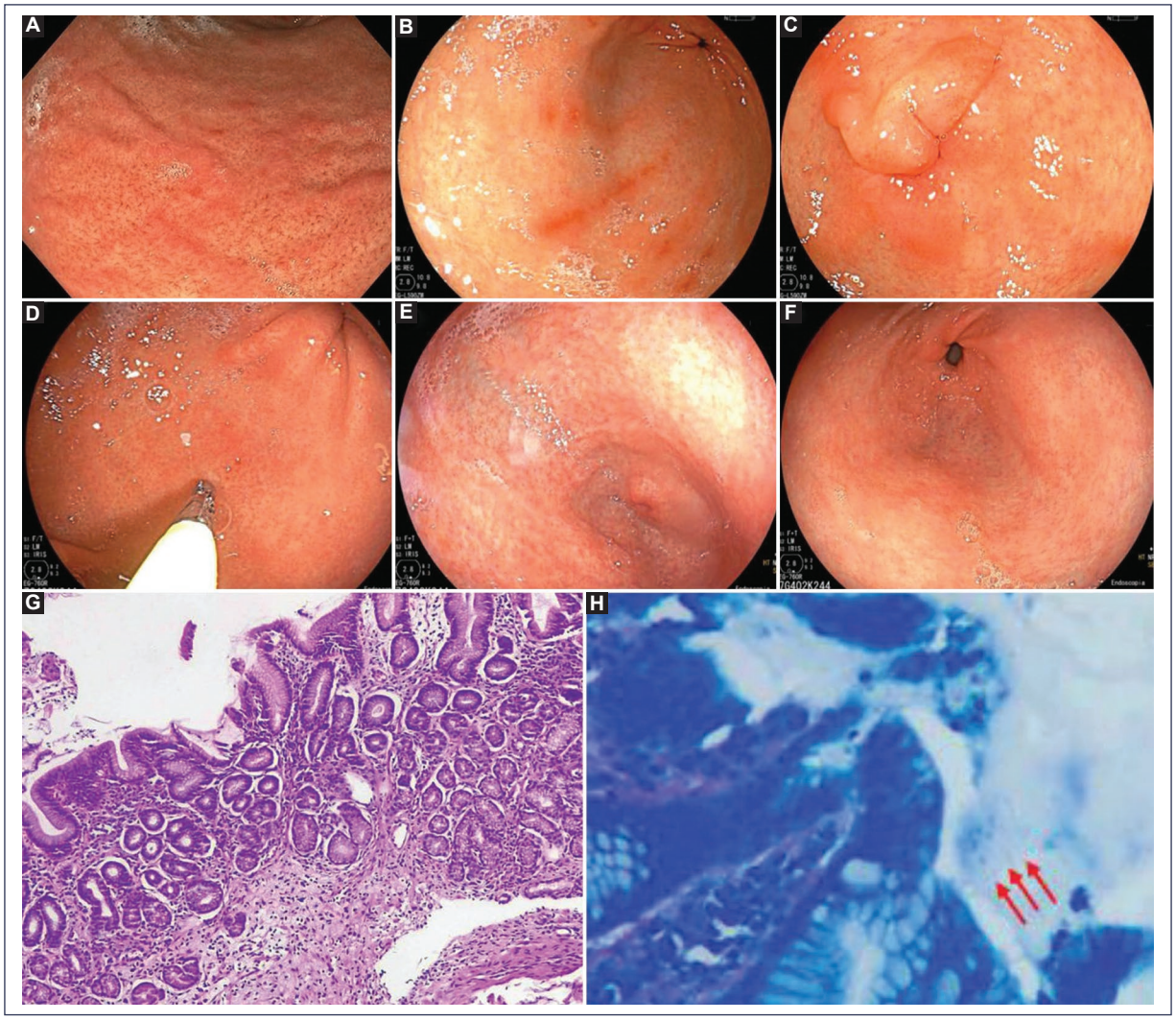

Figure 1. Common endoscopic and histopathological findings in patients with gastritis (*images courtesy of Dr. Genaro Vázquez-Elizondo*). **A:** mucosa with edematous appearance and erythema. **B:** linear erosions in the antrum and body. **C:** prepyloric erythema. **D:** diffuse erythema in the body. **E:** erythema and flattened changes in the antrum and body. **F:** minimal mucosal changes. **G:** mononuclear infiltrate typical of gastritis (hematoxylin and eosin stain). **H:** bacillary structures compatible with *Helicobacter pylori* (Diff-Quick stain). All biopsies from these patients showed the presence of this microorganism (red arrows).

multifactorial – as previously explained – current international and national clinical practice guidelines direct treatment toward specific causes, which typically include eradication of *H. pylori*, reducing or preventing toxicity from drugs such as nonsteroidal anti-inflammatory drugs, and the use of acid-blocking agents such as H2 receptor blockers, proton pump inhibitors, or potassium-competitive acid blockers, according to the specific clinical context.[8,12-16] Additionally, for purely symptomatic control, it is recommended to combine these pharmacological options with general measures, which include suspension of precipitating dietary elements and reviewing the appropriateness and indication of some drugs (such as acetylsalicylic acid and iron, among others).

One of the most frequent considerations when treating patients with gastritis is the eradication of *H. pylori*, which is recommended in those with evidence of atrophic gastritis or intestinal metaplasia,

Table 3. Histopathological findings in gastric biopsy pathology reports

Criterion	Mechanism/relevance	Normal finding	Measurement
Mononuclear leukocyte infiltrate	Intensity of gastritis	2 to 5 lymphocytes, plasma cells, or macrophages per high-power field 2 or 3 cells between foveolae Lymphocytes: < 25/100 epithelial cells	Mild (+) Moderate (++) Severe (+++) Lymphocytes: 25/100
Neutrophil infiltrate	Gastritis activity	2 to 5 neutrophils per high-power field 2 or 3 neutrophils	Location: Lamina propria (+) Epithelium (++) Glandular lumen (+++)
Helicobacter pylori density	Presence (and quantity) of *H. pylori*	Presence or absence	Mild Moderate Severe
Foveolar hyperplasia	Expansion of gastric glands and presence of dysplasia	Description of regenerative changes in glandular epithelium	Difference from dysplastic lesions: Indefinite for dysplasia Atypical foveolar hyperproliferation
Gastric atrophy	Loss of gastric glands (mucosecreting or oxyntic)	Atrophy should not be present Two subtypes: Non-metaplastic: Gland disappearance with sclerosis of lamina propria Metaplastic: Gland replacement: intestinal metaplasia vs. pseudopyloric metaplasia (oxyntic glands)	1 = 1-30% specimens 2 = 31-60% specimens 3 = > 60% specimens
Endocrine cell hyperplasia	Enterochromaffin cell hyperplasia	Hyperplasia and cell alignment should not exist	Precursor of gastric neuroendocrine tumors Alignment of 5 enterochromaffin cells in lamina propria
Lamina propria fibrosis and muscularis mucosae hypertrophy	Scarring following ulcerative or inflammatory processes	Relevant to distinguish from mucosal atrophy	Indicates scarring process
Oxyntic epithelium hypertrophy	Hypertrophic changes in oxyntic epithelium	Usually related to use of proton pump inhibitors and in atrophic gastritis	Report with uncertain clinical significance

Developed from Dixon et al.[3] and Pennelli et al.[19].

Table 4. OLGA reporting system

Global atrophy score		Atrophy score in oxyntic tissue (body and fundus)		
0: no atrophy 1: 1-30% specimens 2: 31-60% specimens 3: > 60% specimens 0		1	2	3
Atrophy score in mucosecreting tissue (antrum and angular incisure)	0	No stage	Stage I	Stage II
	1	Stage I	Stage I	Stage II
	2	Stage II	Stage II	Stage III
	3	Stage III	Stage III	Stage IV

Translated from Pennelli et al.[19].

since it has been shown to stop the progression of neoplastic changes.[17] In patients with dyspeptic symptoms, eradication is a first-line treatment, as it improves the magnitude of symptoms.[8,12] Recently, the Mexican Association of Gastroenterology published the Fifth Mexican Consensus on the Diagnosis and Treatment of *Helicobacter pylori* Infection, in which the guidelines for this therapeutic option can be consulted.[18] Regarding other pharmacological alternatives, there is evidence for the use of mucoprotective agents, acid blockers, and cytoprotective agents. Table 5 summarizes the options available in Mexico.

Table 5. Pharmacological alternatives for the management of gastritis

Drug	Mechanism of action	Type of available studies	Doses evaluated	Clinical outcomes	Comments
Rebamipide	Prostaglandin stimulation, reduction of oxidative stress and modulation of inflammatory cytokines	9 RCTs; 1 prospective study, 1 comparative study with active agent	300 mg/day divided doses for 2 and up to 26 weeks (8 weeks standard)	Improvement in symptom score, endoscopic lesions, and histological inflammation indices	Studied in erosive and chronic gastritis Effect superior to sucralfate and similar to famotidine
Sucralfate	Cytoprotective agent that forms a barrier over damaged mucosa promoting healing through stimulation of prostaglandins and mucus	3 RCTs, 2 placebo-controlled	4 to 6 g/day divided doses (6 and up to 12 weeks)	Histological improvement in chronic non-erosive bile-mediated gastritis post-cholecystectomy), but no effect on dyspeptic symptoms	Borderline effect compared to ranitidine Effect inferior to rebamipide
Famotidine	H2 receptor antagonist in parietal cells	3 RCTs comparative with active compound; 1 escalating dose comparative study	5 to 20 mg/day in different doses for 2 weeks	Non-inferior effect to esomeprazole and rebamipide	Used for short periods and not evaluated beyond 2 weeks
Ranitidine	H2 receptor antagonist in parietal cells	4 comparative RCTs with active compound	300 mg divided doses for 8 weeks; up to 6 months in maintenance	Consistent effect vs. placebo, but inferior to proton pump inhibitors in erosions and histology Superior to sucralfate in symptoms and histology	Effect superior to sucralfate, but less than other acid-suppressing agents
Omeprazole	First-generation proton pump inhibitor	2 comparative and multicenter RCTs in patients using nonsteroidal anti-inflammatory drugs and ulcers or > 10 erosions	20 to 40 mg/day for 4 to 8 weeks; up to 6 months maintenance	Effect superior to misoprostol and ranitidine in erosions at 4 and 8 weeks	Evidence in patients with nonsteroidal anti-inflammatory drug toxicity
Esomeprazole	Second- generation proton pump inhibitor	Comparative RCT with famotidine	10 mg daily for 2 weeks	Effect similar to 20 mg ranitidine	Reduced doses in patients with erosive gastritis
Rabeprazole	Second-generation proton pump inhibitor	3 comparative RCTs in patients with endoscopic and histological gastritis Evaluated in reactive (bile) gastritis	20 mg/day for 4 to 12 weeks	Effect similar to sucralfate in biliary gastritis Effective in chronic non-ulcerative gastritis	Limited evidence and not superior to sucralfate
Tegoprazan	Potassium-competitive acid blocker	2 phase III RCTs compared to placebo	10 to 20 mg/day divided doses for 2 weeks	Consistent effect and superior to placebo for short periods	Still no comparative studies against active compound

RCT: randomized controlled trial.
Developed from Kim et al.[2].

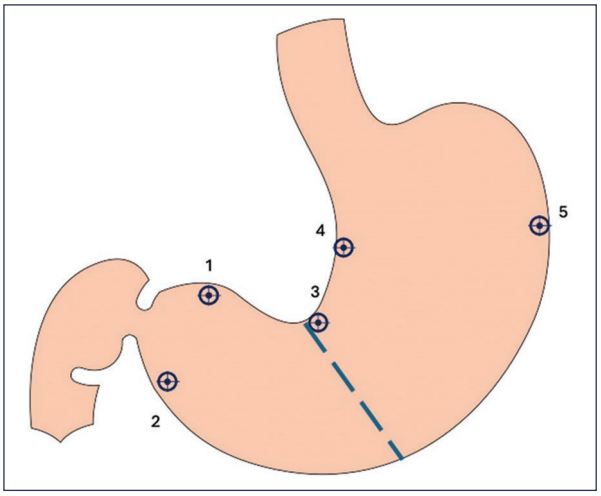

Figure 2. Standard Sydney protocol for biopsy sampling.[3,4]

Conclusions

Erosive gastritis is a clinical-endoscopic condition with multifactorial etiology. The optimal approach integrates: identifying and treating the cause (especially *H. pylori* and exposure to nonsteroidal anti-inflammatory drugs) and suppressing acid and protecting the mucosa when indicated. Acute and chronic management of this condition, including follow-up studies, will largely depend on the specific clinical context.

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Conflicts of interest

The author declares having no conflicts of interest.

Ethical considerations

Protection of human and animal subjects. The author declares that no experiments were performed on humans or animals for this research.

Confidentiality, informed consent, and ethical approval. The study does not involve personal data, medical records, or human biological samples, so ethical approval is not required. SAGER guidelines do not apply.

Declaration on the use of artificial intelligence. The author declares that no type of generative artificial intelligence was used for the writing or creation of content of this manuscript.

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