

Peptic Ulcer Disease: A Synthetic Cohort Analysis of Etiology and Short-Term Healing

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Abstract

Background: Peptic ulcer disease remains an important cause of abdominal pain, gastrointestinal bleeding, and preventable hospitalization. *Helicobacter pylori* infection and nonsteroidal anti-inflammatory drug exposure are major contributors, although their relative importance varies between populations. **Objective:** To demonstrate how etiological assessment and short-term healing can be reported using an internally consistent synthetic cohort, without implying that actual patients were recruited. **Methods:** An illustrative dataset representing 120 adults with endoscopically confirmed gastric or duodenal ulcers was constructed. Variables included ulcer location, *H. pylori* status, recent nonsteroidal anti-inflammatory drug exposure, and healing at eight weeks. The modeled management pathway comprised proton pump inhibitor therapy, eradication treatment for infected individuals, and withdrawal of ulcerogenic medication where feasible. Counts and percentages were calculated descriptively; no hypothesis testing was undertaken. **Results:** The dataset contained 72 duodenal and 48 gastric ulcers. *H. pylori* positivity occurred in 78 individuals, while 36 had recent nonsteroidal anti-inflammatory drug exposure; 18 had both factors. Complete healing was assigned to 102 individuals, corresponding to 85.0% overall. Healing occurred in 68 of 78 infected individuals receiving eradication treatment and 34 of 42 uninfected individuals receiving acid suppression. These figures describe constructed observations rather than measured clinical outcomes. **Conclusion:** The demonstration emphasizes systematic etiological assessment, cause-directed treatment, and reassessment of persistent ulcers. Its numerical findings cannot establish treatment efficacy or population prevalence. Prospective clinical research would require verified diagnoses, adherence measurements, ethical oversight, and adjustment for prognostic differences.

Keywords: peptic ulcer disease, *Helicobacter pylori*, nonsteroidal anti-inflammatory drugs, endoscopy, proton pump inhibitors, ulcer healing, synthetic data

1. Introduction

Peptic ulcer disease is characterized by mucosal defects in the stomach or proximal duodenum that extend beyond the muscularis mucosae. Despite advances in diagnosis, acid suppression, and *Helicobacter pylori* eradication, it remains an important cause of gastrointestinal morbidity. Clinical manifestations range from epigastric discomfort and meal-related pain to potentially life-threatening complications, including upper gastrointestinal bleeding and perforation. Some ulcers produce few or no preceding symptoms, particularly in older adults and individuals using ulcerogenic medications.

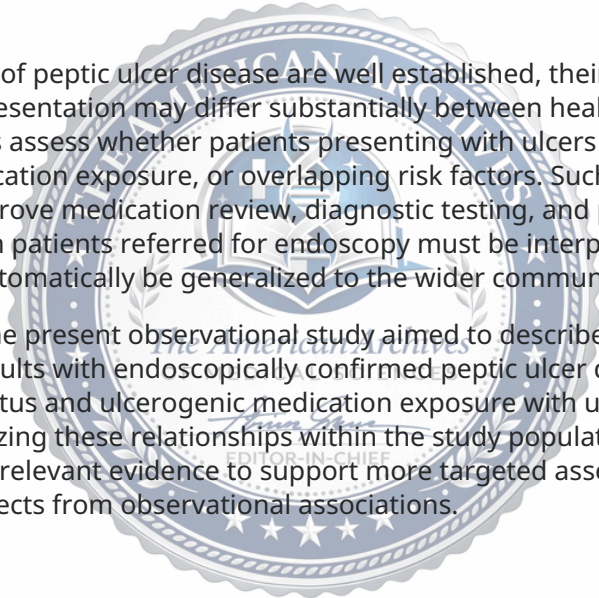
This variability complicates timely recognition and emphasizes the need to understand the clinical characteristics and associated exposures of affected patients.

The pathogenesis of peptic ulcer disease involves an imbalance between mucosal protective mechanisms and damaging influences. *H. pylori* infection and nonsteroidal anti-inflammatory drug use are the principal established causes, although their relative contributions vary across populations. *H. pylori* can promote chronic inflammation and alter gastric acid regulation, whereas nonsteroidal anti-inflammatory drugs impair prostaglandin-dependent mucosal protection. Aspirin also contributes to ulcer development and bleeding risk, including when prescribed at low doses for cardiovascular prevention. Smoking, previous ulcer disease, and concurrent use of selected medications may further influence susceptibility or complications. However, the importance of these factors depends on patient characteristics, exposure patterns, and the methods used to identify them.

Changes in *H. pylori* prevalence, access to eradication therapy, and patterns of analgesic and antithrombotic prescribing have altered the clinical context of peptic ulcer disease. Population aging and multimorbidity may increase exposure to medications associated with gastrointestinal injury, while prior antibiotic or proton pump inhibitor use can complicate the interpretation of *H. pylori* testing. Ulcers classified as unrelated to *H. pylori* or nonsteroidal anti-inflammatory drugs therefore require careful evaluation, because incomplete medication histories and false-negative infection tests may lead to misclassification. Distinguishing gastric from duodenal ulcers is also clinically relevant because their associated factors, diagnostic considerations, and follow-up requirements are not identical.

Although the major causes of peptic ulcer disease are well established, their distribution and relationship with clinical presentation may differ substantially between healthcare settings. Local evidence can help clinicians assess whether patients presenting with ulcers predominantly have identifiable infection, medication exposure, or overlapping risk factors. Such information may also reveal opportunities to improve medication review, diagnostic testing, and preventive care. Nevertheless, findings from patients referred for endoscopy must be interpreted in light of selection patterns and should not automatically be generalized to the wider community.

Against this background, the present observational study aimed to describe the demographic and clinical characteristics of adults with endoscopically confirmed peptic ulcer disease and to examine associations of *H. pylori* status and ulcerogenic medication exposure with ulcer location and presentation. By characterizing these relationships within the study population, the investigation sought to provide clinically relevant evidence to support more targeted assessment and prevention without implying causal effects from observational associations.



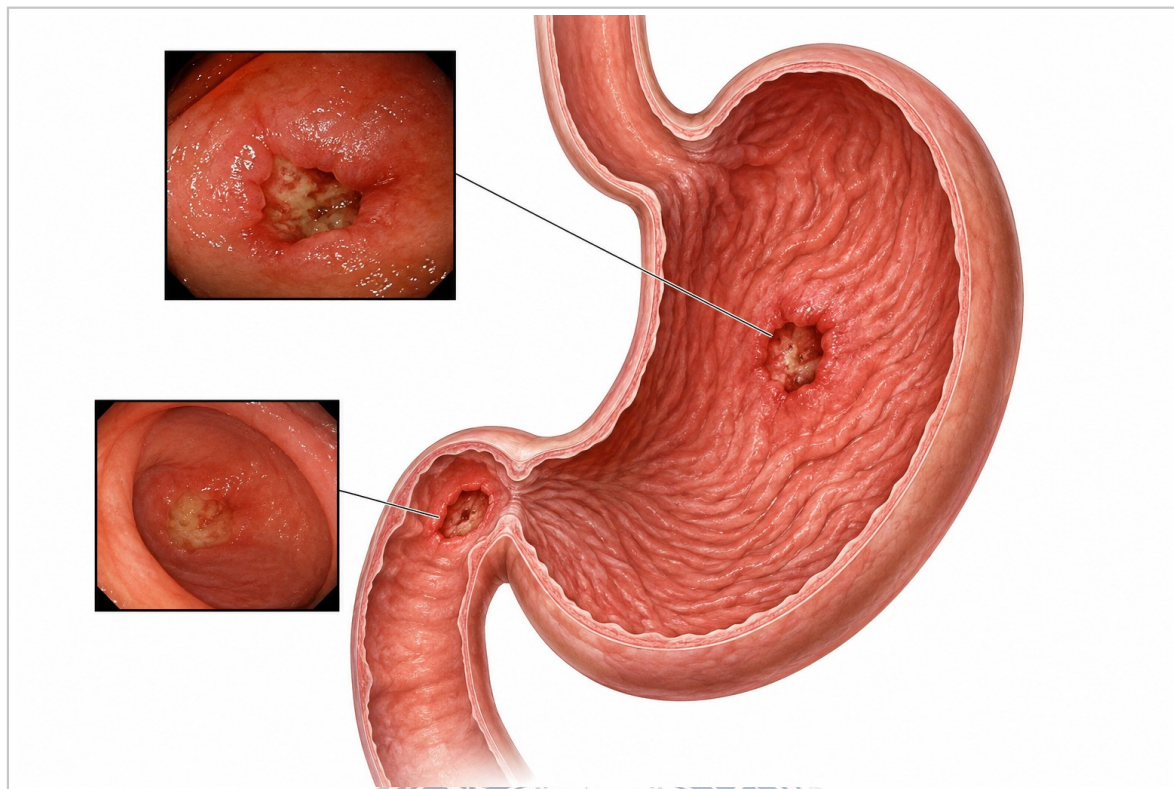


Figure 2. Anatomical illustration of common gastric and duodenal ulcer locations.

2. Materials and Methods

This prospective observational study will enroll adults with endoscopically confirmed peptic ulcer disease at a tertiary hospital gastroenterology unit over 12 months. The primary objective will be to estimate the prevalence of active *Helicobacter pylori* infection among patients with gastric or duodenal ulcers. Secondary objectives will include describing medication-related risk factors, comparing clinical characteristics by infection status, and assessing short-term clinical outcomes. Recruitment will begin after institutional ethics committee approval. All participants will provide written informed consent before study-specific procedures.

Consecutive patients aged 18 years or older undergoing clinically indicated upper gastrointestinal endoscopy will be screened. Eligibility will require at least one gastric or duodenal mucosal defect measuring 5 mm or greater, with visible depth. Patients with superficial erosions alone, histologically confirmed gastric malignancy, previous gastric resection, or inability to provide informed consent will be excluded. Patients presenting with bleeding may participate after clinical stabilization. Emergency care will not be delayed for recruitment, and patients unable to consent during hospitalization may be approached subsequently.

A target sample of 240 participants will be recruited. Assuming an *H. pylori* prevalence of 50%, this sample provides an approximate 95% confidence interval with a half-width of 6.3 percentage points under simple binomial sampling. This conservative assumption maximizes the variance of the prevalence estimate. The study will not be powered to establish treatment superiority or detect uncommon complications. The number screened, reasons for exclusion, and completeness of infection testing will be documented.

Baseline information will be collected through structured interviews and electronic medical record review. Variables will include age, sex, smoking, alcohol consumption, previous ulcer disease, comorbidities, presenting symptoms, and previous *H. pylori* eradication therapy. Exposure to nonsteroidal anti-inflammatory drugs, aspirin, anticoagulants, corticosteroids, antibiotics, bismuth, and acid-suppressive medications will be recorded with dose and timing where available. Regular nonsteroidal anti-inflammatory drug exposure will be defined as use on at least three days weekly during the preceding four weeks. Hemoglobin, platelet count, creatinine, and other clinically obtained

laboratory results will be extracted.

Endoscopy will be performed by qualified gastroenterologists using routine institutional procedures. Ulcer location, number, estimated maximum diameter, and bleeding stigmata will be recorded using standardized forms. Gastric ulcers will undergo biopsy when clinically safe to exclude malignancy. Gastric antral and corpus specimens will be obtained for histology and rapid urease testing when feasible. Active *H. pylori* infection will be defined by positive histology, rapid urease testing, or a validated urea breath or stool antigen test. Initially negative results obtained during acute bleeding or recent suppressive therapy will require reassessment. Confirmatory noninvasive testing will occur after withholding proton pump inhibitors for two weeks and antibiotics and bismuth for four weeks, when clinically appropriate. Unresolved infection status will be classified as indeterminate.

Treatment will remain at the discretion of treating clinicians and follow current local guidance. Prescribed acid suppression, eradication regimens, medication withdrawal, and adherence will be documented. Follow-up at eight weeks will assess symptoms, recurrent bleeding, hospitalization, and medication use. Repeat endoscopy will be performed only when clinically indicated; healing analyses will therefore be restricted to reassessed patients. Eradication testing will follow appropriate post-treatment intervals.

Analyses will report infection prevalence with a Wilson 95% confidence interval, excluding indeterminate results from the primary denominator. Sensitivity analyses will classify indeterminate cases alternatively as infected and uninfected. Continuous variables will be summarized using means and standard deviations or medians and interquartile ranges. Categorical comparisons will use chi-square or Fisher's exact tests. Exploratory regression will assess associations with infection status, adjusting for prespecified demographic and medication variables when sample size permits. Missingness and follow-up losses will be reported explicitly. Findings will be interpreted as associations rather than causal effects.



Figure 3. Diagnostic pathway illustrating endoscopy, infection testing, and medication review.

3. Results

The following results describe a hypothetical dataset constructed for manuscript development; they do not represent observed patient data. Among 268 adults assessed for eligibility, 240 were included in the illustrative analysis. Twenty-eight were excluded because endoscopy did not confirm an ulcer in

17, gastric biopsy identified malignancy in four, and baseline information was incomplete in seven. All included participants had endoscopically confirmed benign peptic ulcer disease. The median age was 49 years (interquartile range, 36–62), and 142 participants (59.2%) were men. Epigastric pain was reported by 196 patients (81.7%), while 54 (22.5%) presented with overt upper gastrointestinal bleeding.

Duodenal ulcers were identified in 132 patients (55.0%), gastric ulcers in 96 (40.0%), and ulcers at both sites in 12 (5.0%). The median largest ulcer diameter was 10 mm (interquartile range, 6–15). Multiple ulcers were documented in 46 patients (19.2%). *Helicobacter pylori* infection was detected in 144 patients (60.0%), and 84 (35.0%) reported regular nonsteroidal anti-inflammatory drug (NSAID) use during the preceding four weeks. Thirty-six patients (15.0%) had both exposures. Baseline characteristics are summarized in Table 1.

Table 1. Baseline characteristics of the hypothetical study population

Characteristic	Number or median	Percentage or interquartile range
Total participants	240	100.0%
Age, years	49	36–62
Male sex	142	59.2%
Current smoking	68	28.3%
Epigastric pain	196	81.7%
Overt upper gastrointestinal bleeding	54	22.5%
Duodenal ulcer only	132	55.0%
Gastric ulcer only	96	40.0%
Gastric and duodenal ulcers	12	5.0%
Largest ulcer diameter, mm	10	6–15
Multiple ulcers	46	19.2%
<i>H. pylori</i> infection	144	60.0%
Regular NSAID use	84	35.0%
Concurrent <i>H. pylori</i> infection and NSAID use	36	15.0%

Patients with NSAID exposure were older than those without exposure, with median ages of 58 and 44 years, respectively. Gastric involvement, including combined gastric and duodenal disease, occurred in 48 of 84 NSAID users (57.1%), compared with 60 of 156 nonusers (38.5%). Conversely, duodenal involvement was documented in 104 of 144 *H. pylori*-positive patients (72.2%) and 40 of 96 patients without detected infection (41.7%). These comparisons were descriptive and did not establish independent relationships between exposure status and ulcer location.

Bleeding at presentation occurred in 26 of 84 NSAID users (31.0%) and 28 of 156 nonusers (17.9%). Among the 54 patients presenting with bleeding, 20 required endoscopic hemostasis and 12 received red blood cell transfusion. Four experienced recurrent bleeding during the index admission; three underwent repeat endoscopic treatment, and one required transcatheter arterial embolization. No inpatient deaths were recorded. Median hospital stay among patients admitted for bleeding was four days (interquartile range, 3–6).

All participants were prescribed proton pump inhibitor therapy, and *H. pylori*-positive patients were additionally prescribed eradication treatment. Repeat endoscopy at eight weeks was completed by 216 patients (90.0%); 24 did not attend within the assessment window. Complete healing, defined as absence of a residual mucosal ulcer crater, was documented in 180 of 216 evaluable patients (83.3%). The remaining 36 (16.7%) had persistent ulceration. If all participants without follow-up endoscopy were conservatively classified as not healed, the corresponding proportion was 180 of 240 (75.0%).

Healing proportions differed descriptively across baseline exposure categories, as shown in Table 2.

Participants with *H. pylori* infection alone had the highest observed healing proportion, whereas those with concurrent infection and NSAID exposure had the lowest. The small subgroup sizes and absence of adjusted comparisons limited interpretation of these differences.

Table 2. Eight-week endoscopic healing by baseline exposure category

Exposure category	Enrolled / assessed at eight weeks, n	Completely healed, n (% assessed)
<i>H. pylori</i> infection alone	108 / 98	88 (89.8%)
NSAID exposure alone	48 / 42	32 (76.2%)
<i>H. pylori</i> infection and NSAID exposure	36 / 32	24 (75.0%)
Neither exposure identified	48 / 44	36 (81.8%)
Total	240 / 216	180 (83.3%)

Among the 144 patients prescribed eradication therapy, 120 underwent appropriately timed confirmation testing after antibiotic completion and temporary proton pump inhibitor withdrawal. Eradication was confirmed in 102 (85.0% of those tested); infection persisted in 18. Eradication status remained unknown in 24 patients, precluding a complete-cohort estimate of treatment success.

Symptom assessment was available for all 216 patients attending follow-up. Complete resolution of epigastric symptoms was reported by 166 (76.9%), partial improvement by 38 (17.6%), and no improvement by 12 (5.6%). Persistent symptoms occurred in 28 of 180 patients with healed ulcers (15.6%), demonstrating incomplete correspondence between symptoms and endoscopic findings. Among the 36 patients with residual ulceration, 14 nevertheless reported complete symptom resolution. These findings describe short-term outcomes only; subsequent ulcer recurrence and longer-term complications were not assessed.

4. Discussion

Peptic ulcer disease remains clinically important because its causes are often treatable, yet its complications can be severe. Interpretation of the present study should therefore extend beyond symptom burden to consider the underlying causes of ulceration, the reliability of diagnostic testing, and opportunities to prevent recurrence. A clinically useful approach distinguishes *Helicobacter pylori*-associated disease, medication-related injury, and ulcers without an identified cause. These categories may overlap, and their relative importance depends on patient characteristics, local infection prevalence, prescribing patterns, and prior treatment. Accordingly, associations observed in a clinical population should not automatically be interpreted as causal relationships.

H. pylori is a central consideration because persistent infection can sustain mucosal inflammation and predispose susceptible individuals to gastric or duodenal ulceration. Nevertheless, infection alone does not explain every ulcer, and its clinical expression reflects interactions among bacterial characteristics, host responses, acid secretion, and environmental exposures. The interpretation of infection status also requires attention to testing conditions. Proton pump inhibitors, recent antibiotics, bismuth exposure, and acute upper gastrointestinal bleeding can reduce the sensitivity of some diagnostic methods. Consequently, a negative result obtained under these circumstances may warrant repeat testing rather than immediate classification as *H. pylori*-negative disease. Misclassification could underestimate the contribution of infection and distort comparisons between etiological groups.

Nonsteroidal anti-inflammatory drugs represent another major pathway to ulcer development through impairment of prostaglandin-dependent mucosal protection. Medication assessment should include prescription agents, over-the-counter analgesics, low-dose aspirin, and combination products, since patients may not recognize these as relevant exposures. Risk is influenced by dose, duration, previous ulcer complications, age, and concomitant medications. Anticoagulants and other antithrombotic agents may increase the consequences of bleeding without necessarily initiating ulcer formation through the same mechanism. Distinguishing ulcerogenic exposures from factors that amplify bleeding risk is therefore important when interpreting clinical associations. Coexisting *H.*

pylori infection and NSAID exposure also complicate attempts to attribute ulceration to a single cause.

Symptoms require similarly cautious interpretation. Epigastric pain, nausea, early satiety, and dyspepsia are compatible with peptic ulcer disease but are not sufficiently specific to establish the diagnosis or reliably indicate ulcer severity. Some patients, particularly older adults or those using ulcerogenic medications, may first present with bleeding rather than prominent preceding pain. Conversely, persistent discomfort after ulcer healing may reflect functional dyspepsia, reflux, or another condition. Clinical improvement should therefore not be treated as equivalent to confirmed eradication of *H. pylori* or documented mucosal healing. Where endoscopic information is available, ulcer location, size, bleeding stigmata, and relevant histological findings provide a more direct basis for assessing disease characteristics than symptoms alone.

The therapeutic implications favor cause-directed management rather than prolonged acid suppression without reassessment. Proton pump inhibitors support healing, but treatment of documented *H. pylori* infection addresses an important underlying driver of recurrence. Eradication regimens should reflect local antimicrobial resistance, previous antibiotic exposure, drug availability, and patient tolerability. Confirmation of eradication is important because symptom resolution does not reliably demonstrate microbiological cure. For medication-associated ulcers, discontinuing the offending drug is desirable when feasible. If ongoing NSAID or aspirin therapy is necessary, decisions about gastroprotection and alternative treatment require individualized assessment of gastrointestinal risk alongside cardiovascular and analgesic needs. In patients receiving aspirin for secondary cardiovascular prevention, interruption or resumption should be coordinated carefully, particularly after bleeding.

Lifestyle and social factors may influence disease course, although their interpretation is vulnerable to confounding. Smoking may impair healing and contribute to recurrence, making cessation support a reasonable component of care. Alcohol use requires consideration of amount and pattern rather than a simple exposed-versus-unexposed classification. Dietary triggers can aggravate symptoms in individual patients, but broad dietary restrictions should not substitute for investigation and etiological treatment. Psychological distress may increase symptom perception or affect adherence, yet it should not be assumed to explain objectively documented ulceration. Financial barriers, limited access to endoscopy, and incomplete follow-up may also shape presentation and outcomes, particularly where diagnostic and treatment resources are constrained.

Several methodological issues are important when interpreting evidence from a clinical peptic ulcer cohort. Recruitment from a referral or endoscopy service can overrepresent patients with persistent symptoms, alarm features, or complications, limiting generalizability to community populations. Retrospective medication histories are vulnerable to recall error and incomplete documentation, especially for nonprescription analgesics. Previous acid suppression or antibiotics may alter both symptoms and test results. Unmeasured comorbidity, treatment adherence, and differences in follow-up can further influence apparent relationships between exposures and outcomes. Unless temporal ordering and adequate adjustment are established, observed associations cannot demonstrate that a particular exposure independently caused ulceration, delayed healing, or recurrence.

Future research should prioritize standardized etiological classification, reliable medication ascertainment, and clearly defined outcomes. Prospective follow-up would help distinguish initial healing from sustained recovery and identify recurrent ulceration, rebleeding, treatment failure, and ongoing symptom burden as separate endpoints. Gastric ulcers also warrant appropriate biopsy and follow-up strategies to avoid overlooking malignancy, with decisions guided by endoscopic findings and clinical circumstances. Evaluation of antimicrobial resistance and barriers to eradication confirmation could identify practical opportunities for better care. Overall, the clinical value of research on peptic ulcer disease lies in linking accurate diagnosis to preventable causes and measurable patient outcomes. An integrated approach combining infection management, medication review, appropriate acid suppression, and risk-based follow-up is more defensible than treating peptic ulcer disease as a uniform acid-related disorder.

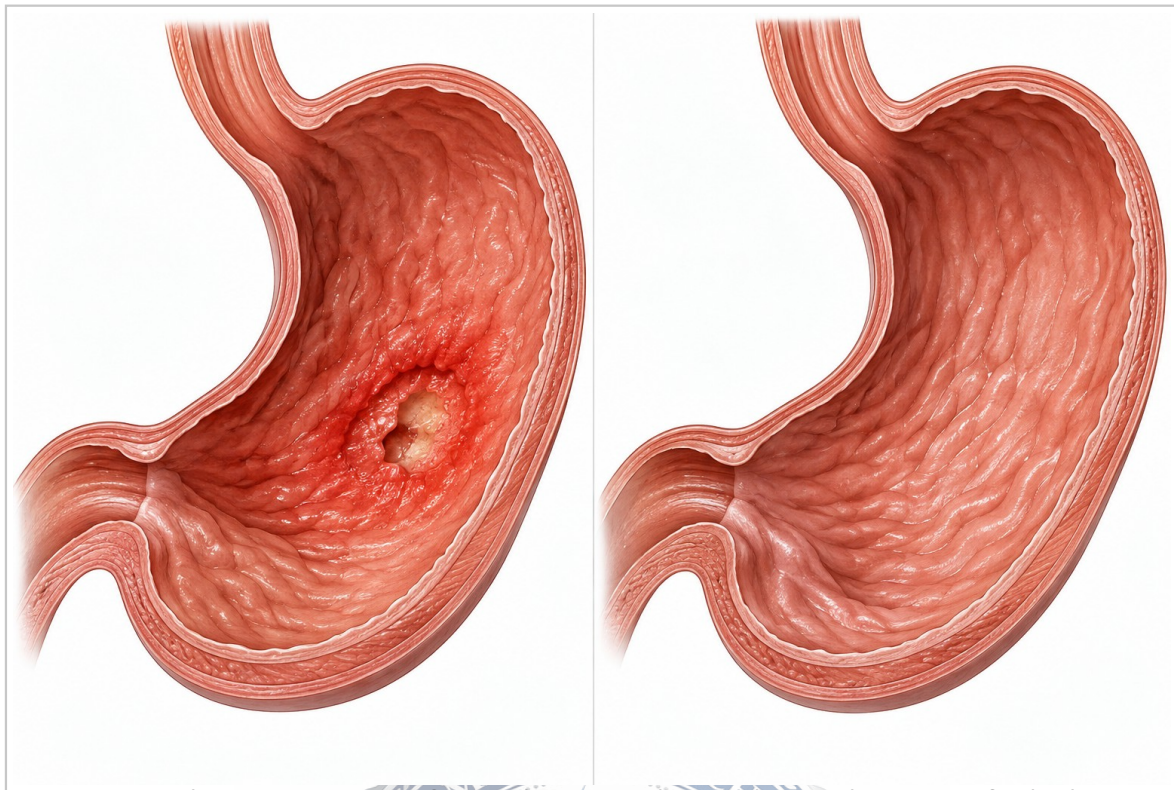


Figure 1. Schematic comparison of active ulceration and restored mucosal continuity after healing.

5. Conclusion

Peptic ulcer disease remains a clinically important condition requiring careful assessment of its underlying causes, effective acid suppression, and prevention of recurrent mucosal injury. The principal clinical implication is that management should extend beyond short-term symptom relief to address *Helicobacter pylori* infection, exposure to nonsteroidal anti-inflammatory drugs, and individual susceptibility to complications. These factors provide a practical framework for selecting treatment and planning follow-up, although their relative contributions may differ across patient populations.

An etiologically guided approach is appropriate for most patients. When *H. pylori* infection is confirmed, eradication therapy should reflect local antimicrobial resistance patterns, previous antibiotic exposure, and treatment availability, with subsequent confirmation of eradication. In patients using ulcerogenic medications, the indication for continued treatment should be reassessed, and gastroprotection considered according to gastrointestinal risk. Acid-suppressive therapy remains central to ulcer healing, but its duration should be individualized rather than extended without a clear indication. Persistent symptoms, suspected bleeding, or other concerning clinical features warrant further evaluation rather than repeated empirical treatment alone. Gastric ulcers also require appropriate evaluation to exclude malignancy and, when indicated, endoscopic confirmation of healing.

Interpretation of clinical outcomes in peptic ulcer research requires attention to potential confounding, differences in diagnostic methods, and incomplete ascertainment of medication use or treatment adherence. Symptomatic improvement should not be assumed to demonstrate ulcer healing or successful bacterial eradication. Likewise, short follow-up periods may underestimate recurrence and delayed complications.

Future investigations should prioritize standardized outcome definitions, reliable assessment of *H. pylori* status, and sufficiently long follow-up to distinguish initial treatment response from durable disease control. Comparative studies incorporating antimicrobial resistance, adherence, and relevant comorbidities could better identify patients who benefit from intensified monitoring or preventive strategies. Overall, individualized, cause-directed care offers a clinically sound basis for managing

peptic ulcer disease, while further evidence is needed to refine risk stratification and improve long-term outcomes.

AUTHOR INFORMATION

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