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Stomach Ulcers: A Silent Digestive Disorder — A Comprehensive Review

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ABSTRACT :

Gastric and duodenal ulceration together form peptic ulcer disease (PUD), a condition that continues to affect large numbers of people worldwide despite receiving comparatively little public attention. This article draws together published evidence on how common the disease is, how it develops, how it is diagnosed, and how it is treated, with particular focus on two dominant causes: infection with *Helicobacter pylori* and regular use of nonsteroidal anti-inflammatory drugs (NSAIDs). It also looks at treatment approaches recommended in current clinical guidelines — chiefly proton pump inhibitors (PPIs) and antibiotic eradication regimens — along with newer strategies aimed at limiting serious complications like bleeding and perforation. Taken together, the evidence points to one clear message: catching the disease early and addressing its risk factors proactively is what ultimately keeps complication rates and healthcare costs down.

Keywords: *Peptic ulcer disease, gastric ulcer, Helicobacter pylori, NSAIDs, proton pump inhibitors, gastrointestinal bleeding*

1. Introduction

A stomach ulcer is essentially a break in the lining of the digestive tract deep enough to pass through the muscularis mucosae — the thin muscle layer beneath the surface epithelium — most often located in the stomach itself or in the upper part of the duodenum. For a long time, these ulcers were blamed largely on stress and lifestyle. That view has since been overturned: the two factors now understood to drive most cases are *H. pylori* infection and long-term NSAID use. What makes the disease particularly tricky is that roughly two out of every three cases produce no noticeable symptoms until something goes wrong [1].

Because so many ulcers stay quiet until they bleed or perforate, diagnosis frequently comes late — often only after a complication has already developed. This article works through the published evidence with that problem in mind, pulling together epidemiological findings and the guidance issued by major clinical bodies into a single, readable overview for students, clinicians, and researchers alike [2].

2. Literature Review

The scale of the problem is considerable: current reviews put the annual global case count in the millions, a figure explained mainly by how common *H. pylori* infection is and by how routinely NSAIDs are used, whether prescribed or bought over the counter [3]. Mechanistically, *H. pylori* burrows into the gastric mucus layer and interferes with the stomach's normal defenses, setting off a chronic inflammatory response. NSAIDs work through a different route entirely — by blocking prostaglandin synthesis, they cut down the stomach's own protective output of mucus and bicarbonate.

What is striking is how these two mechanisms compound one another. A patient with both an active *H. pylori* infection and regular NSAID use faces up to six times the ordinary ulcer risk, and their bleeding risk climbs even higher than that. Work carried out in and around Jaipur has added to this picture, showing that the underlying disease mechanisms are not identical across different population groups [4].

On the treatment side, the Japanese Society of Gastroenterology's 2021 guidelines favor PPI-based triple therapy as the standard first-line option for clearing *H. pylori*, while flagging vonoprazan as a newer agent worth watching. Where NSAIDs are the culprit, the usual response is either to stop the drug altogether or, if that is not possible, to add a PPI alongside it. Active bleeding is treated as an emergency — rapid fluid resuscitation followed by endoscopy — with eradication confirmed only afterward. A smaller subset of ulcers fit neither the *H. pylori* nor the NSAID pattern; these tend to stem from severe physiological stress or, less commonly, malignancy, and need to be worked up individually [5].

3. Methodology

This is a narrative review built on peer-reviewed material published between 2010 and 2025, drawn from PubMed, PubMed Central, and leading gastroenterology journals. Searches combined terms such as "peptic ulcer disease," "gastric ulcer," "*H. pylori*," "NSAID-induced ulcer," and "PUD

management" [6].

Preference was given to randomized controlled trials and cohort studies wherever they existed, and to sources that aligned with currently active clinical guidelines. Papers were included if they focused on adult patients and reported outcomes relevant to complications. From each source, information was pulled on disease prevalence, risk factors, the diagnostic tools in use — endoscopy and breath testing chief among them — and how well PPIs, antibiotics, and endoscopic procedures actually perform in practice [7].

4. Discussion

At its core, PUD comes down to a tug-of-war between damaging forces — acid, pepsin, NSAIDs — and the stomach's own defenses, namely mucus, bicarbonate, and adequate blood flow to the tissue. *H. pylori* tips that balance further by way of its urease enzyme and a set of cytotoxins that directly injure the mucosa [8]. When the balance fails, the result is a break in the lining that classically causes gnawing epigastric pain after meals, though, as noted earlier, many patients feel nothing at all.

Endoscopy remains the gold standard for confirming a diagnosis, especially where a patient is high-risk or shows warning signs, while the urea breath test offers a useful non-invasive alternative [9]. Treatment centers on wiping out *H. pylori* using PPI-triple therapy — typically amoxicillin plus clarithromycin — which brings recurrence down from roughly half of patients to almost none. For NSAID-related cases, stopping the drug or covering it with a PPI is the standard approach, and guidelines increasingly mention vonoprazan as a preventive option as well [10].

When bleeding does occur, the priority is endoscopic control of the bleed combined with intravenous PPI therapy, followed later by a test to confirm eradication was successful. Two areas worth watching going forward are vonoprazan, which suppresses acid more powerfully and for longer than older PPIs, and probiotics as a possible aid to mucosal repair — though the evidence base for the latter is still thin and developing [11].

5. Results

Pulling the numbers together: PUD is not confined to any one age group, *H. pylori* prevalence runs as high as 50% in certain regions, and NSAIDs appear to account for somewhere between a fifth and a third of all cases [12]. PPI-triple therapy clears the infection successfully in 80–90% of patients, which in turn speeds healing and cuts perforation risk by around 70%.

Patients with both an NSAID-related ulcer and concurrent *H. pylori* infection face a 3.5- to 6.5-fold jump in complication risk compared with either factor alone [13]. For bleeding ulcers specifically, endoscopic treatment stops the bleed in about 9 out of 10 cases, and of the roughly 10% that rebleed, most can still be managed with extended PPI therapy [14].

Looking at who is affected, incidence appears higher among African American and Hispanic populations and among people with a first-degree relative who has had PUD — a pattern that points toward both genetic susceptibility and socioeconomic influences. Silent, symptom-free ulcers make up roughly a third of the total caseload, which is itself a strong argument for screening patients who fall into recognized high-risk groups rather than waiting for symptoms to appear [15].

Table 1. Key Figures from the Reviewed Literature

Parameter	Reported Value
Cases with no symptoms at diagnosis	~66%
<i>H. pylori</i> prevalence (highest regions)	Up to 50%
Cases attributable to NSAID use	20–30%
PPI-triple therapy eradication success	80–90%
Drop in perforation risk after eradication	~70%
Risk increase, combined <i>H. pylori</i> + NSAID use	Up to 6-fold
Endoscopic hemostasis success rate	~90%
Rebleeding rate post-hemostasis	~10%
Recurrence after successful eradication	Falls from 50–60% to near 0%

6. Conclusion

Stomach ulcers may develop quietly, but their impact is anything but minor — *H. pylori* and NSAIDs remain the two factors clinicians need to address together, following an integrated, guideline-based approach [16]. Picking up the disease early through endoscopy or breath testing, then following through with proper eradication therapy and sensible NSAID management, is what actually moves the needle on complications and recurrence [17].

Looking ahead, there is room for more work on personalized treatment strategies, on whether probiotics genuinely help, and on making eradication therapy more accessible in parts of the world where resources are limited. This review is offered as a practical, up-to-date reference for anyone — clinician, researcher, or student — working to understand where PUD management currently stands [18].

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